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In The Matter Of:

Norma R. Broin, et al. v.
Philip Morris Companies, Inc., et al.

John D. Spengler, Ph.D.
Vol. 1, April 30, 1997

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Volume I
Pages 1 to 272
Exhibits 1-8

IN THE CIRCUIT COURT OF THE 11TH JUDICIAL CIRCUIT
IN AND FOR DADE COUNTY, FLORIDA
GENERAL JURISDICTION DIVISION

NORMA R. BROWN, et al., :
Plaintiffs, :
vs. : Case No.
: 91-49738 CAZZ
PHILIP MORRIS COMPANIES, INC., :
et al., :
Defendants. :

DEPOSITION OF JOHN D. SPENGLER, Ph.D., a witness called on behalf of the Defendant R.J. Reynolds Tobacco Company, taken pursuant to Rules 1.310 and 1.390 of the Florida Rules of Civil Procedure, before Anne H. Bohan, Registered Diplomate Reporter and Notary Public in and for the Commonwealth of Massachusetts, at the Offices of Goodwin, Procter & Hoar, Exchange Place, 53 State Street, Boston, Massachusetts, on Wednesday, April 30, 1997, commencing at 10:10 a.m.

PRESENT:

Stanley M. Rosenblatt, Esq., P.A.
(by John A. Hoag, Esq.) Concord Building
66 West Flagler Street, Miami, FL 33130,
for the Plaintiffs.

Coll Davidson Carter Smith Salter & Barkett P.A.
(by Barry R. Davidson, Esq.)
3200 Miami Center, 201 South Biscayne
Boulevard, Miami, FL 33131-2312,
for the Defendants Philip Morris
Incorporated and Philip Morris Products
Inc.

PRESENT, Continued:
Womble Carlyle Sandridge & Rice
(by Jeffrey L. Furr, Esq. and Kimberly C. Stevens, Esq.) 200 West Second Street,
Post Office Drawer 84, Winston-Salem, NC
27102, for the Defendant R.J. Reynolds
Tobacco Company.

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PROCEEDINGS

JOHN D. SPENGLER, Ph.D.
a witness called for examination by counsel for the Defendant RJ. Reynolds Tobacco Company, being first duly sworn, was examined and testified as follows:

DIRECT EXAMINATION
BY MR. FURR:

Q: Good morning, Dr. Spengler.
A: Good morning.
Q: As I just introduced myself to you off the record, my name is Jeff Furr. I represent the R.J. Reynolds Tobacco Company, and I'll be the first person to ask you questions today.
Do you understand that your deposition is being taken in a case that is referred to as the "Broin case" that's pending in the state court in Miami?
A: Yes, I do.
Q: Have you had your deposition taken before?
A: Not in this case, but in other cases.
Q: In other cases. And so are you generally familiar with how depositions are conducted?
A: Yes, I am.
Q: You understand that there will be a series

of questions and answers that will be recorded by the court reporter?
A: Yes.
Q: And you understand that you've been placed under oath just as you would be if you were testifying in court?
A: Yes.
Q: Just a few basic rules that I think would help us both today. If you don't understand my question or need it repeated for any reason, please just indicate so, because I want to make sure that you understand the question. And second, I would ask you not to speculate but to respond factually, and if you are having to draw inferences from the facts that you are aware of, let us know when that process has occurred. Is that acceptable?
A: Yes.
Q: Finally, we all fall into this sometimes, including myself. The court reporter can only record verbal responses, so attempt to respond verbally as opposed to nodding your head, if you can remember, please.
Dr. Spengler, how did you get involved in this case?

A: I was called by the law firm Stanley Rosenblatt.
Q: Do you recall when you first heard from Mr. Rosenblatt?
A: Approximately a month and a half, two months ago.
Q: And when Mr. Rosenblatt contacted you, what did he tell you?
A: He asked me if I'd be willing to testify in this particular case, and he explained briefly what the case was about.
Q: And what did he tell you about the case?
A: He told me that it was a case where he represented the plaintiffs in a class action suit, which then included 60,000 flight attendants, against the tobacco industry.
Q: Did Mr. Rosenblatt make any specific request of you?
A: No.
Q: What work have you performed in relation to your involvement in this case?
A: I reviewed, and not comprehensively or thoroughly, the documents that were sent to me. And I just briefly last night went over parts of the

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National Academy document on airliner cabin environment.
Q: So you've been sent some documents with respect to this case?
A: Yes, I have.
Q: And who did you get the documents from?
A: The law firm.
Q: Did you bring those documents with you today?
A: No, I did not.
Q: Can you describe those documents for us?
A: Yes.
MR. FURR: Let me interrupt. If Mr. Hoag wants to represent those are the bound documents that have been distributed to all the witnesses in the case, we'll be happy to accept that.
MR. HOAG: The 42 document index. If there's anything else he's familiar with – I'm sure that's what he's primarily referring to.
THE WITNESS: Yes.
A: And then I was also sent Dr. Burns' deposition and a book which I did not read.
Q: Have you reviewed Dr. Burns' deposition?
A: About two thirds of it.

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Q: Do you recall the title of the book that you were sent?
A: Cigarette Papers or Tobacco Papers. You know the one I'm referring to.
Q: Are you being compensated for your work in this case?
A: Yes, I am.
Q: And what rate are you being compensated at?
A: $250 an hour.
Q: How many hours have you worked on the case?
A: About five.
Q: Do you have further work that you need to do in preparation for testifying in this case?
A: That's not clear to me.
Q: What do you mean when you say that's not clear to you?
A: Not knowing what's anticipated in terms of my detailed familiarity with other people's depositions, there may be some additional work. I don't plan to do any measurements, I don't plan to do any calculations, but that remains to be seen.
Q: Have you – you told us that you had reviewed Dr. Burns' deposition. Have you reviewed any other depositions?

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A: No, I have not.
Q: Have you spoken to other experts involved in the case?
A: Not since being retained.
Q: Who did you speak to prior to being retained?
A: Well, I know several. I know Don Stedman, and I know Dr. Burns, and I worked with them in the past.
Q: Have you spoken to other experts regarding the case?
A: No.
Q: Dr. Spengler, have you been designated as an expert witness in any other litigation involving tobacco?
A: No.
Q: Have you testified before as an expert?
A: Yes.
Q: What types of litigation have you testified in?
A: Litigations involving indoor air pollution cases, office buildings in particular, and hazardous waste sites.
Q: Let's start with the indoor air pollution

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cases. How many times have you testified in those cases?
A: By that you mean testified in court as opposed to depositions?
Q: Let's start with testifying in court, and then we'll go to depositions.
A: There were two cases that actually went to trial.
Q: Where were those cases tried?
A: Here in Massachusetts.
Q: Taking them one at a time, can you tell me what those cases were about?
A: Yes. One case was a plaintiff's claim against the Honeywell Corporation.
Q: What type of claim was it?
A: It was a plaintiff injury claim that a defective electrostatic air cleaner caused the production of ozone and subsequent effects on his wife or to his wife.
Q: And what was the nature of your testimony?
A: In that case I worked on behalf of Honeywell, and we had done many measurements of ozone indoors, as well as we examined the emission rates from electrostatic precipitators, Honeywell

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models in particular, and determined that the levels claimed could not have been achieved.
Q: In that case did you express opinions regarding the health implications of ozone exposure?
A: I may have, but that wasn't central to what I did.
Q: Do you have a copy of the transcript of your testimony in that case?
A: I personally don't, no.
Q: What was the second case that you testified in?
A: The second case was –
Q: Excuse me, the second indoor air case.
A: That's what I assumed. That case involved a homeowner who was claiming that fumes from a leaky underground storage tank, gasoline storage tank, were penetrating into his basement.
Q: On whose behalf did you testify?
A: It was on behalf of the defense, the gasoline owner, the station owner.
Q: And what was the nature of your testimony in that case?
A: After essentially making a site visit and determining the kinds of sources that were in the

person's house, in the garage, without any direct measurements that would differentiate emissions that were coming from the gasoline storage tank leak, one couldn't determine whether indeed they had penetrated into the house.
Q: What were the substances at issue in that case?
A: It was primarily benzene and other gasoline hydrocarbons.
Q: Did you express any opinions in that case regarding the health implications of the exposures?
A: No, I did not.
Q: Do you have a transcript of your testimony in that case?
A: No, I don't.
Q: Have you also been deposed in other indoor air cases?
A: Yes.
Q: Do you recall how many?
A: There are two others that I recall.
Q: But you did not testify at trial in those cases?
A: No, I did not.
Q: Have those cases been completed, to your

knowledge?
A: One has, and the other I don't know the status of.
Q: Taking them again one at a time, could you tell me about the two indoor air cases that you were deposed in.
A: Yes. The more recent one had to do with a building in Cambridge, Massachusetts, that the plaintiff alleged that they were constructively evicted because of indoor air-quality-related issues, without going into detail, it gets complicated. So you can ask if you want to follow up.
Q: Sure. On whose behalf did you testify on in that case?
A: The –
Q: Excuse me. Who named you as an expert in that case?
A: The company that managed the property.
Q: What was the substance of the testimony that you gave in your deposition?
A: It really was responding to questions related to a whole series of measurements that we had made during the investigation of the building.

Q: And what substances were those measurements made of?
A: The substances included biological components such as fungal spores, fibers, volatile organic compounds, and various engineering measurements.
Q: Was tobacco smoke an issue in that case?
A: No, it was not.
Q: Do you have a copy of your deposition transcript?
A: Somewhere. It probably resides in the files at my company.
Q: I'm sorry, the files of what?
A: At my company.
Q: What company is that?
A: Environmental Health and Engineering.
Q: Is that the company through which you do your consulting?
A: Yes, it is.
Q: Was there another indoor air case that you were deposed in?
A: Yes.
Q: Could you tell us about that case.
A: This had to do with a building in

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California. It goes back quite a while; let me see if I can recall some of the details. It was an action taken by some of the employees against the company that owned the building, so they both worked in the building for the company that had essentially built the building.
Q: And were you named as an expert in that case?
A: Yes, I was.
Q: And which party were you an expert for?
A: For the plaintiffs.
Q: Can you tell us the nature of the testimony you gave in your deposition in that case?
A: Yes. I was asked to review various industrial hygiene documents that were prepared by other consultants who had examined the building while the alleged exposures were occurring.
Q: And what were the exposures at issue in that case?
A: The best that I could recollect or determine – not recollect, determine – from the documents that I saw, it related to a variety of compounds that may be emitted in higher concentration during remodeling and buildout of the

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property. And again, this is recollection and probably in the area of speculation, I guess. I think there were some issues having to do with the early operations of the ventilation system.
Q: What were the sources – do you recall what the sources of the emissions were in the case?
A: I believe that they related to sort of conventional furnishings and coating materials and ceiling compounds that you might find on construction sites.
Q: Was tobacco smoke at issue in the case?
A: No, it was not. Just for clarification, as I'm recounting this I recall a third case, just for completeness.
Q: Could you tell us about that third case.
A: There was a building in Alaska, in Anchorage, Alaska, and it was rented by the state, and eventually at the recommendation of the state epidemiologist, the building was closed until fixes could be made.
Q: And what was your role in that case, in the Alaska case?
A: Yes. We had done a site visit, done some measurements of that building, but our role was

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primarily to review work done by the state industrial hygienist and to review building documents and building diagrams on how the building operated.
Q: Was tobacco smoke at issue in that case?
A: It could have contributed, but it didn't come up in any of our measurements because the building was empty by the time we were there.
Q: Were you deposed in the case, in the Alaska case?
A: Yes, I was.
Q: During your deposition did you express any opinions on tobacco smoke contamination?
A: No, I didn't, that I recall. This goes back many years now.
Q: Do you have a copy of the transcript of your deposition in the Alaska case?
A: No, I don't.
Q: I don't think I asked you this: Do you have a copy of the transcript of your deposition in the California case?
A: No, I don't.
Q: You also said that you've testified in some cases involving hazardous waste.

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A: Yes.
Q: Was that trial or deposition testimony?
A: In one case it was both, and in the other case it was just deposition.
Q: Did either of those cases involve tobacco smoke?
A: No.
Q: Do you have transcripts of your depositions in those cases?
A: Again, they may reside in our long-term storage files at the company.
Q: Where were those cases located?
A: One was in New York, Buffalo, New York, it was heard in Federal Court, and the other was in San Diego. Excuse me, I take that back. The deposition was in San Diego; the site in question was in Riverside County.
Q: In Riverside?
A: Yes.
Q: That's an area that you've done some research in, isn't it?
A: Yes, it is.
Q: Have you ever expressed opinions regarding tobacco smoke contamination in indoor environments

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in any litigation prior to today?
A: No.
Q: And that would include trial testimony and deposition testimony?
A: Yes, it would.
Q: And you're not currently working on any other tobacco-related litigation; is that correct?
A: That's correct.
Q: Could you review for us, or identify for us first, I should ask, the Congressional testimony that you have given related to environmental tobacco smoke in indoor environments including airline cabins. And when I ask you to identify, I just mean, to your recollection, the committee that you testified before and the time frame as opposed to what you actually said at this point.
A: I haven't thought about it since it occurred, but I do recall that myself, my other vice-chair of that committee, and the chair of that committee and maybe others, did testify before Congress. I have no recollection of whether it was House or Senate or what subcommittee it was in front of.
Q: Was this the Subcommittee on Aviation

testimony?
A: It could have been. I've testified more recently to a committee looking into aviation health and safety issues.
Q: Let's –
A: If you can tell me the time frame, I can...
Q: I'm actually interested in any Congressional testimony that you've ever given on a number of subjects beginning with tobacco smoke. And your recollection is that you did testify one time on issues related to environmental tobacco smoke?
A: No. Well, it wasn't that specifically. I tell you, I am rather sure that members of the committee, myself included, did testify about the findings of the committee, of which tobacco smoke was a component. I could be corrected on that; I mean, there were press hearings and there were several other things going on. But I do not ever recall going to Congress to specifically talk about tobacco smoke in any circumstance. It's one of several indoor pollutants, and it may have been mentioned in testimony that I've made about indoor

air pollution too.
Q: A couple of times you have referred to the committee's findings in responding to the last question. Which committee are you talking about?
A: I was talking about the Committee on Airline Cabin Environment, and that is a committee assembled by the National Research Council.
Q: Do you have copies of any Congressional testimony that you've given?
A: I must somewhere in my files.
(An interruption)
Q: Dr. Spengler, did you do anything in particular to prepare for your deposition today?
A: Other than what I mentioned before, no.
Q: Did you meet with Mr. Hoag this morning?
A: Yes, I did.
Q: And what did you-all discuss?
A: In fact, briefly some of the things we just talked about, whether I had given deposition before or testified before, but he didn't pursue any details as you have. He asked me if I knew some of the other individuals that he had recently deposed or have been deposed in this case. I didn't. I had a chance to review only in part some of the

materials that were prepared by the Environmental Protection Agency in response to their document about passive cigarette smoke effects, lung cancer and respiratory effects, and so forth. And that was a multi-page document; I just read the part on epidemiology.
Q: And is that a document that Mr. Hoag provided to you this morning?
A: Yes, it is.
Q: Have you ever reviewed the full "Environmental Protection Agency Report on Environmental Tobacco Smoke and Health"?
A: I've read parts of it but did not review it as an outside reviewer.
Q: Was there anything else that you and Mr. Hoag talked about?
A: I asked him if he knew who would be taking the deposition, and he couldn't be sure.
Q: Anything else?
A: I'm trying to think. Oh, yes. We talked about – I asked, actually, what the defense's strategy was, and he related to me what he thought it might be.
Q: And what was that?

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A: Well, you know it better than I do.
Q: I'm open to enlighten myself here.
MR. DAVIDSON: We would just like to hear it from another source, that's all.
A: Just that in these cases that the issue is to obfuscate and to say there may be lots of contributing causal factors, and that it would be difficult to identify one specific cause, and that's the gist of the strategy which we talked about.
Q: Was there anything else specifically that he told you?
A: We talked about the airplane environment and other things that I might know about. So ask your question now.
Q: You know a lot about other things in the airplane environment, don't you, Dr. Spengler?
A: I know a few things, yes.
Q: It's been one of your areas of interest for some time, hasn't it?
A: Yes, it has.
Q: Dr. Spengler, I'm not going to mark this at this point, but I'm going to hand you a document that was provided to us by plaintiffs as a copy of your curriculum vitae and ask you to take a look at

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it briefly, please.
A: (Witness reviews document) Actually, as I look at it, I do recall another indoor air pollution case that I have given deposition for. I'm sorry, it wasn't fresh in my mind. And so if you wanted to go back to that.
Q: Sure, we can stop it now.
A: It had nothing to do with tobacco smoke.
Q: You expressed no opinions in this other case that you recollect regarding tobacco smoke; is that correct?
A: That's correct.
Q: As you look at that, I'll tell you that my first question is, is there anything else that you would add to that CV to bring it up to date?
A: Well, what made me think of this other case was that it was pointed out to me in my previous deposition that I had somehow forgot to list one of my earlier publications that had to do with exposures, not tobacco, in enclosed ice skating rinks. It's probably not relevant to this case, but that was missing from the resume by mistake.
Q: And what types of exposures did you address in the ice skating rinks in the publications not

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present in your CV?
A: Carbon monoxide exposure.
There are some things that have been published since this was prepared.
Q: Can you identify those for us.
A: I was an editor along with Richard Wilson on a recent publication entitled Particles in our Air: Concentrations and Health Effects published in late 1996 and distributed through Harvard University Press.
Q: Did that involve – did that publication involve indoor air?
A: Yes, it was discussed in some of the chapters.
Q: Did this publication report on original research you had conducted, or was it in the nature of a review?
A: The book included a broad range of topics related primarily to concentrations, composition, exposure and health effects of outdoor air pollution.
Q: Did you discuss tobacco smoke in the book?
A: One of the chapters, not one that I wrote, did refer to tobacco smoke.

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Q: Who wrote that chapter?
A: John Evans and Scott Wolff.
Q: And who are they?
A: John Evans is a faculty member at the Harvard School of Public Health.
Q: And what issues involving tobacco smoke did they address in that chapter?
A: They had proposed a lung damage model that could be used to explain how some health outcomes could be observed due to exposures to outdoor air pollutants, and part of that model was derived from loss of lung function observed in smoking populations.
Q: Okay. Were there other publications that you would add to your CV to bring it up to date?
A: There were some other recent ones but not having to do with particles or tobacco smoke.
Q: I'm going to ask you to keep a copy of the curriculum vitae with you and use it if it's helpful in any way to respond to the next series of questions.
Dr. Spengler, you have done a great deal of research regarding the measurement of particles in indoor environments; is that correct?

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A: Yes.
Q: And that research addresses the attribution of indoor particles to tobacco smoke in part, doesn't it?
A: In part, that's correct.
Q: Using your CV if it's helpful, could you describe for us, as comprehensively as you can, and again, I'm just asking you to identify, the individual projects that you have been involved with measuring particles in indoor air including environmental tobacco smoke articles.
A: Yes. I can attempt to do that.
Q: Can you do that for us.
A: I stand corrected. This is updated; it did include the publication I just referred to, sorry, Wilson and Spengler, editors.
Q: Dr. Spengler, just so I make sure that you understand my question, now I'm asking you to identify for me research you have done. If there are publications related to that research, I'd appreciate you pointing those out also, but I want to focus on research, regardless of whether you have published on it or not. Okay?
A: Correct. Let me use my resume --

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Q: Sure.
A: -- as a guide to that. And this is going chronologically backwards. We participated in the particle exposure study that was conducted in Riverside, California, and its predecessor, a pilot study that was done in -- recent in Southern California in and around the towns of Glendora and Azusa.
Q: And what was your role in that study?
A: This was a collaborative study between myself and my colleagues and staff and researchers at the Research Triangle Institute, and there were other collaborators. My specific role was in the planning, the implementation and the analysis of that study.
Q: And how did it relate to tobacco smoke?
A: Well, it related to tobacco smoke because we were attempting to identify the sources that may contribute to personal exposures to particulate matter and wanted to make sure we could correctly attribute that particulate mass to several of the primary sources, which included tobacco smoke.
Q: So in that study did you make determinations with respect to the contribution of

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environmental tobacco smoke to indoor particulate levels?
A: Yes. In the analysis we made a calculation as to how much of the exposures the people received may have come from other people's cigarette smoke.
Q: And have you published those results?
A: Yes.
Q: Is that publication listed in your CV?
A: Yes. The results should be included in a publication with the lead author my staff assistant, Haluk Ozkaynak, published in 1996. And it should also be referred to in two other papers, one with the lead author Thomas published in 1993 and Clayton published in 1993.
Q: All three of those publications relate to the same study?
A: Yes.
Q: Who is the custodian of the data collected in that study?
A: The Environmental Protection Agency.
Q: Do you have a copy of the data that you have access to?
A: Yes, yes. This is in the public domain too.

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Q: The data are in the public domain? How would I go about obtaining a copy of the data and any supporting documentation I would need to analyze the data?
A: The entire study is described in a multivolume set, and it has an EPA publication number on it. And I don't know who's responsible for distributing the diskettes with the data in the electronic format, but it would either be the principal investigator of the prime contract, and that would be a Dr. Edo Pellizzari of the Research Triangle Institute, or the principal staff scientist for the Environmental Protection Agency, which would be Dr. Lance Wallace.
Q: But your understanding is that that data is in the public domain now?
A: Yes, it is.
Q: What's the next study, research project that you've been involved with that is related to tobacco smoke in indoor environments?
A: There were a series of publications that came out of our study on air pollution health effects that is commonly referred to as the Six Cities study because six communities were involved

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in the study.
Q: Has that study been completed?
A: The actual field data collection has been completed, yes, some time ago.
MR. HOAG: What number is that? That's on Page 16? Which study is it or studies?
Q: Dr. Spengler, is it fair to say there in fact have been a whole series of publications related to the Harvard Six Cities?
A: Yes, that is true.
Q: And what has your role in that study been?
A: I have acted as a coinvestigator, coprincipal investigator, and at the time was responsible for the collection of the air pollution data and the analysis or joint analysis of the air pollution data.
Q: And how does that data relate to environmental tobacco smoke in indoor environments?
A: Only to the extent that particles measured in indoor environments also included various source contributions such as cooking and outdoor pollution, as well as combustion indoors, including cigarette smoke.
Q: So that data can be used to derive

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estimations of the contribution of tobacco smoke to particles in the indoor environment?
A: Yes.
Q: And in fact, you have reported on that in some of the publications, haven't you?
A: Yes.
Q: Who is the custodian of that data, Dr. Spengler?
A: That would be Dr. Douglas Dockery.
Q: Do you have access to the data?
A: I guess I could, yes.
Q: Do you have a copy of the data in your custody now?
A: No, I don't.
Q: Are there other projects related – research projects related to tobacco smoke that you've performed?
A: Let me say, by way of generality, that all studies in which we measured personal exposures to particles would have included exposures to particles generated by tobacco smoke.
Q: Other than the ones that you already identified, how many additional studies like that are there that you've been involved with?

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A: Almost all of them have been under the aegis of the Six City study, and so they became subcomponents of those, with the possible exception of some studies conducted in the early 1980s, late 1970s, with funding from the Environmental Protection Agency.
Q: Doctor, is it fair to say that you rely upon the studies that you've been describing as a basis for your opinions regarding environmental tobacco smoke exposure in indoor environments?
A: No, not entirely, no.
Q: But in part?
A: Along with a body of literature that has been published by other investigators, yes.
Q: You do believe that these studies that you have described have contributed to your understanding of environmental tobacco smoke exposure in indoor environments, don't you?
A: Let me answer it this way: These studies have helped us to understand the contribution of a variety of sources to both indoor levels of particulate matter, as well as personal levels of particulate matter, yes.
Q: And that would include the contribution of

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tobacco smoke to indoor and personal particulate matter exposures?
A: I'm not sure I follow your question. It seems a bit circular. So why don't you ask it again.
Q: Sure. You said that these studies have contributed to your understanding of a variety of sources of indoor air exposures; is that correct?
A: Correct.
Q: And my question is, tobacco smoke would be one of those sources that you have come to understand better through these studies; is that correct?
A: You just want to put a fine point on it.
Q: Right.
A: Fine, yes.
Q: Are there any other research projects on environmental tobacco smoke exposures that you've been involved with that we haven't discussed yet?
A: Two others come to mind.
Q: Could you identify them for us, please.
A: We assisted our coauthors and coinvestigators in measurements of particles in restaurants that had partial separation of smoking

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and nonsmoking sections.
Q: Is there a publication in your CV related to that project?
A: I believe there should be. Yes, there is.
Q: And which publication is that?
A: Its lead author is Lambert on Page 17.
Q: Okay. Is there another project? I'm sorry, let me follow up on this. Who has custody of the data collected in that study, Dr. Spengler, the Lambert study?
A: I guess Lambert would.
Q: Do you have a copy of the data?
A: No, I don't.
Q: Could you identify the second additional ETS exposure research that you recollect about.
A: There was a study that was done, it must be at least ten years ago or so. I'm trying to find the reference that would describe that. It had to do with a survey of office buildings in the Boston area.
Q: Was something in fact published on that survey?
A: Yes, there was. I found it now on Page 14. The lead author was Miesner. The fourth

reference down.
Q: Dr. Spengler, do you have possession, either personally or in your company or in your academic department, of any data sets that contain the raw data that has been conducted in any of the research that you've described?
A: No, I don't.
Q: Let's now talk about your research, your original research –
MR. HOAG: Could we take a break? I need to call my office.
MR. FURR: Sure.
(Recess)

BY MR. FURR:

Q: Dr. Spengler, my first question is, did you meet with Mr. Hoag during the break?
A: Yes.
Q: And what-all did you discuss?
A: We discussed essentially where you were heading with all these questions. That's what we talked about.
Q: And what did Mr. Hoag tell you?
A: He said that they have it all in front of them and they'll eventually get to these articles

and we'll get to talk about them.
Q: Earlier today you told us that you asked Mr. Hoag what the Defendants' strategy in this case was. Why was that of interest to you?
A: I've been involved in a number of cases, and maybe I'm just curious about legal positioning and what I thought were areas that you might be probing into.
Q: Did you discuss those areas with Mr. Hoag?
A: Yes, I think we did.
Q: And what were those areas?
A: The areas were radiation and ozone.
Q: And why did you think those were areas that we might probe into?
A: I don't think I thought that. I said in his experience in other depositions that he was listening to or taking part in, those areas had come up.
Q: We have been talking about the original research you have done on that would include the ability to assess ETS exposures indoors. Have you done any original research regarding the health implications of those exposures?
A: No.

Q: Would you now describe for us as comprehensively – I should say identify for us as comprehensively as you can all of the original research that you have participated in involving the airliner cabin. Again, use your CV if that's of help to you.
A: For a number of years out of my own curiosity I used to carry around portable sampling equipment to make my own measurements for my own information, and that probably was in or around the time that the National Research Council committee was meeting.
Q: Was this part of a formal study or just something you did when you flew?
A: It's something I did when I flew. In the last few years we were contracted by 20/20, ABC News, to make measurements on a series of flight segments, domestic flights. And then more recently we had completed a comparison study among various modes of public conveyance, which included airplanes, trains, buses and subway systems.
Q: Beginning with your personal study in which you carried around some portable sampling equipment, did you ever publish the results of those

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measurements?
A: No, I did not.
Q: Do you still have those data?
A: No, I don't.
Q: What happened to them?
A: These were digital readout equipment, and I would just make handwritten notes of the concentrations that I was measuring and never kept those notes.
Q: Did not keep them.
A: No.
Q: With respect to the 20/20, ABC News, research project, have you published a scientific article on the data collected in that project?
A: Not yet.
Q: Do you intend to?
A: Yes.
Q: Is it a work in progress now?
A: Yes.
Q: You do have possession of those data, don't you?
A: Yes.
Q: Would you be willing to produce those for us to look at?

A: No.
Q: Why not?
A: We have yet to publish the results, and I want to make sure that we have the opportunity to publish original research and submit it through a peer review process.
Q: With respect to the comparative study of various modes of public –
A: Conveyances.
Q: – conveyances, I take it those public conveyances included airliner cabins?
A: Specifically, the 777.
Q: Did you obtain original data on the 777?
A: Yes.
Q: Do you have possession of those data?
A: Yes.
Q: Would you be willing to produce those data to us?
A: Not yet.
Q: What type of data did you obtain on the 777?
A: We obtained measurements of carbon dioxide, carbon monoxide, temperature, relative humidity, pressure, occasional measurements of noise and

particulates, and we did additional measurements of volatile organic compounds and biological samples, both airborne and in dust collected from seats.
Q: Who funded that research?
A: Boeing.
Q: What did you find with respect to particulates?
A: I found the levels were very low.
Q: And what were the levels that you found, in an arithmetic mean, first of all?
A: I can't remember what we calculated for arithmetic mean. I can tell you the range of values that I can recollect.
Q: Okay.
A: On the order of 5 to 30 micrograms per cubic meter.
Q: How many samples were taken?
A: There were – I believe there were six flight segments. I do not think we measured particles on the last two segments, so probably measurements on about four segments.
Q: So four flight segments on one type of aircraft?
A: Correct.

Q: Did you simultaneously assess the ventilation rates on that aircraft?
A: No, we didn't.
Q: What method or analytical process did you use to measure the particulates?
A: We used a TSI dust trap, which is an optical particle sampler.
Q: Did you say that, as you sit here, you can't recall what the arithmetic mean of the particulate measurements would have been?
A: The way we typically operate that instrument, we put it on a one-minute averaging sequence. You might sample every second, but then it keeps a one-minute average. So we get a record of one-minute values and plot the trace. We could go back and recalculate what the arithmetic mean was, but at this point we haven't done that.
Q: Now, this was done for Boeing; is that correct?
A: That's correct.
Q: And why did Boeing want this done, or I should ask you, what were you told about why Boeing wanted this done?
A: Well, we made the proposal. The proposal

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was to develop a set of comparable measurements in various modes of transportation, so that the levels of all of these parameters measured on airplanes could be put into context.

Q: How did you select Boeing as the recipient of your proposal?

A: In discussions with them they said that they would entertain such a proposal.

Q: How much did the study cost to conduct?

A: About $70,000 to $80,000.

Q: When was the study performed?

A: Beginning last May of 1996, with the last set of measurements made in December of '96.

Q: Why did you think this study would be of interest to Boeing?

A: There are several reasons, but there was a Subcommittee on Aviation Ventilation set up by the ASHRAE, American Society for Heating, Refrigeration and Air Conditioning Engineers, and that committee was charged with both defining research needs as well as what special ventilation requirements might be necessary for airplanes. And so it was in part – strike that. Boeing as well as a number of other companies had members serving on

that subcommittee. So it was in part of trying to provide additional information to that subcommittee and to Boeing and to the industry at large that we undertook this study.

Q: You said there were several reasons why you felt Boeing might be interested in the study. Were there additional reasons other than those that you just described?

A: Boeing had – let me strike that too. I don't know what Boeing's stance was. There's been a continuing series of complaints among flight attendants made through their unions about the quality of environments on airplanes.

Q: How do you know that?

A: They told me.

Q: I'm sorry, who's told you?

A: I've heard it by direct testimony before Congress, and I've heard it from talking to people in the union and talking to flight attendants who were active in the union.

Q: I'm sorry I interrupted you. Please go ahead.

A: Well, that being a backdrop, Boeing as a manufacturer of aircraft equipment I think would

logically be concerned about the design issues relevant to health and comfort and safety, as well as obviously energy and other considerations of their aircraft.

Q: Did anyone at Boeing ever express those concerns to you?

A: Not in so many words, no.

Q: Did they express these concerns generally to you?

A: Probably the best way to say it is, Boeing has a new research initiative that has just started to explore the possibilities of improving the comfort to passengers and flight crew on airplanes.

Q: Is this initiative also related to health concerns for passengers and crew?

A: As far as I know, there was –

MR. HOAG: Objection. Unless he has it in his personal knowledge, I object to his ability to answer –

MR. FURR: That's been an ongoing agreement. I think the Doctor understands that.

MR. HOAG: Well, his answer implies that he didn't have personal knowledge; that's why I'm objecting.

Q: Go ahead, Doctor.

A: I assume from what I know that it includes all aspects related to the interior environment of aircraft, which would include health, as well as comfort, as well as safety.

Q: Did Boeing fund the study as you proposed it, or did they make any changes in the study?

A: I can't recall in detail. We did discuss the content, and there were some modifications made.

Q: And what were those modifications?

A: I think – I don't recall in detail, because there was sort of a dollar limit, so we had to make choices on how many samples that we could make and where we could make it. I think it resulted that we, to meet our budget constraints, did most of our measurements of the ground transportation in the Eastern Corridor. And they also wanted measurements made in the public transit system in Los Angeles, and we said, "Sure, we can do that," because one of the flight routes of the 777 goes to L.A.

Q: Did you propose to Boeing that you measure contaminants or parameters other than those that you ended up measuring?

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A: I don't quite follow that.
Q: Let me ask it a different way. When you made your proposal to Boeing, did you propose that ozone be measured aboard aircraft?
A: Good point. In that list I should also include that we made ozone and nitrogen dioxide measurements.
Q: I'm sorry, did you say nitrogen dioxide?
A: Yes.
Q: Did you propose to Boeing that radiation be measured aboard the flights?
A: No.
Q: Why not?
A: We really had our hands full with all these parameters, and I wasn't – well, I just couldn't handle another parameter.
Q: What did you find with respect to your ozone measurements?
A: We found that the levels were nondetectable in all except very few samples. They were nondetectable on the airplane samples.
Q: You were not able to measure ozone on the airplane samples?
A: Correct.

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Q: Did that surprise you?
A: No.
Q: What did you find with respect to VOCs?
A: We identified some 20 to 30 compounds or quantified them above detection level. I almost would need the data in front of me in order to be able to say the specific findings. I can tell you in general, some things were higher on airplanes and some things were higher on trains and some things were higher on buses. In fact, I think only three compounds were higher on airplanes than on other modes of transportation.
Q: What were those compounds?
A: They were isopropyl alcohol, acetone and ethyl alcohol.
Q: Have you reported the results of this study to Boeing?
A: A draft report was given to them a week ago.
Q: Would you produce that report to us?
A: No.
Q: Why not?
A: Because we're under contract with Boeing, and they have to have a chance to receive the report

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and comment on the report and have us respond to it.
Q: What were the components of that draft report?
A: The components included the summary findings of some but not all of those parameters that I mentioned, because we haven't completed the analysis for others. And we provided the traces for all the continuous parameters that had been measured for all 26 transportation segments and a description of methods used.
Q: Did the report contain a discussion section?
A: No, not yet.
Q: Is one being developed?
A: It will be.
Q: Did the report contain any discussion of the implications of your findings for the comfort and health of passengers or crew?
A: That will be included in a discussion in an interpretation section.
Q: That section has not been drafted yet?
A: It has not been drafted.
Q: Is it a work in progress?
A: I hope so.

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Q: I understand you've submitted the draft report. Has your written report – have you also reported verbally to Boeing officials about the study?
A: Yes.
Q: In those reports, have you discussed with Boeing officials the comfort and health implications of your findings for passengers or crew?
A: In part, yes.
Q: And what have you told them?
A: Well, I told them two things I guess that would relate to your question. One is about the carbon dioxide levels, which ranged between – let's just put it this way to say less than 1,000 parts per million on fresh air systems once through ventilation systems, to 1,500 or so with planes with ventilation systems that included recirculation, and levels higher than that during boarding periods.
So in the process of trying to interpret what those carbon dioxide levels might mean, I showed them some data that had been recently reported on investigations of health symptoms and performance among schoolchildren in comparison to the carbon dioxide levels measured in their

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classrooms. And we used that by way of trying to interpret whether they were in a range where cognitive function might be altered or symptoms may be altered.

Q: I'm sorry, I didn't mean to interrupt. And what were your conclusions in that regard?

A: We didn't draw any conclusions, only to point out that this study that I was making reference to was the only one that we could find that demonstrated effects at concentration levels in the 1,500 to 5,000 parts per million range, which is below the established occupational level.

Q: And can you identify the reference article that you compared your measurements to with more specificity?

A: I can't recall the lead author's name. It was a set of Swedish – excuse me – Norwegian investigators that reported in the international conference that was held in Nagoya, Japan, in July of 1996.

Q: Do you have a copy of that report?

A: Yes.

Q: Would you produce it to us?

A: Sure.

Q: Okay. What was Boeing's reaction to that information?

A: I don't recall any specific reaction.

Q: You said you reported two things to them, and you identified the CO_2. What was the second?

A: The second one had to do with the perception of acceptability of air and its relationship to temperature and humidity and how that acceptability would then be modified with the additional presence of sources of odor.

Q: And what did you report to them in that regard?

A: I reported to them what looked like sort of the fundamental findings of that investigation which was done in Denmark indicated that at lower entropy, which in this case would be lower relative humidities, that the introduction of odorous sources of volatile organic compounds would be perceived as degrading the acceptability of the indoor environmental air. I also pointed out that these investigations covered a range of conditions that were likely to be found in office buildings but did not extend down to the range of conditions that were encountered on airplanes.

Q: Has Boeing indicated to you what they intend to do with this information?

A: No, I don't. I can say they certainly have inquired about intention to publish it and encouraged us to do so.

Q: Who was the project officer at Boeing that you dealt with?

A: William Spann.

Q: How high did the CO_2 concentrations go during boarding?

A: 2,000 to 3,000 parts per million, and of course it varied.

Q: What are the increased measurements of CO_2 during boarding attributable to, the sources?

A: I think the sources are primarily the same and were the same as during cruise operation; namely, the CO_2 exhaled by people, which would be passengers and crew, and the fact that during the predeparture, ventilation rates are typically lower than during operation.

Q: You've identified the two issues that you reported on the Boeing and told us that the written report regarding the comfort and health implications of your findings is still being drafted; is that

correct?

A: Correct.

Q: As you sit here, have you in your mind concluded what types of points that you intend to make in that written report that you haven't discussed already?

A: Other than the two that I mentioned earlier that we have verbally reported?

Q: Yes, sir.

A: We will have some conclusions and discussions to relate concerning the biological measurements. But as I sit here today, I don't know what we're going to be able to conclude from those measurements yet, only because my colleague, who is an expert in microbiology, and I haven't sat down to discuss the results.

Q: Is that Dr. Burge?

A: That's correct.

Q: Anything else?

A: No.

MR. FURR: Excuse me one minute.

(A pause)

Q: Dr. Spengler, you identified three original research projects on the airliner cabin environment

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for us. Are there any others that come to your
mind?
A: Yes.
Q: And could you describe those for us, could
you identify them for us.
A: Yes. There was a study of ozone
concentrations on airplanes.
Q: Who funded that study?
A: Nobody.
Q: When was it conducted?
A: About three years ago.
MR. HOAG: Which one are we talking about
now, ozone concentrations? Which study is that?
THE WITNESS: It's not listed.
Q: Well, would you describe the study for us,
please.
A: Yes. A student in my night class that I
taught at Harvard was also a flight attendant, and
she wanted to do a class project that was required
of all graduate students, so she chose to measure
ozone on a series of flights that she was working
on.
Q: Have those results been published?
A: No, they have not.

Q: Will they be published?
A: I hope so; I hope they will. It's our
intention to write them up and publish them.
Q: How many flights did she sample on?
A: It was close to 100.
Q: And what type of aircraft are involved?
A: I think they were all wide bodies.
Q: Does "wide body" mean that they had more
than one aisle in the cabin section?
A: Yes. And I believe most of them were 747s;
I'll have to go back and look at the records.
Q: And what routes did she make the
measurements on?
A: Transpacific routes and a few domestic
routes.
Q: Can you describe what she found, the
measurements.
A: She found elevated ozone on some of the
flights. By "elevated" I mean the flight averaged
value, and that average might be three hours in
duration up to 12 hours in duration, exceeded 100
parts per billion.
Q: Exceeded 100 parts per billion.
What were the highest measurements she

found?
A: I believe they were in the high 100s, maybe
200, up to 200 parts per billion.
Q: Do you recall what percentage of the
flights that she sampled exceeded 100 parts per
billion?
A: Something on the order of 15 percent.
Q: Do you recall the arithmetic mean of her
measurements?
A: No.
Q: Who has these data, Dr. Spengler?
A: I do.
Q: Would you be willing to produce them to us?
A: Not immediately, no.
Q: And why is that?
A: Because I'm planning to write this up with
the person who did the study.
Q: What was her name?
A: Susan Ludwig.
Q: Who does she fly for?
A: I'd rather not say. Do I have to say?
MR. DAVIDSON: Yes.
A: I do? Well, just for her sake. Do I have
to answer that?

Q: Well, I'd like for you to. I can't make
you answer it, but there's no basis for –
A: Because when we write it up, we're not
going to attribute it to any particular airlines,
only to say that this is an issue that has to be
looked at.
Q: Well, who does she fly for?
A: Northwest, KLM.
Q: Did Northwest know that this study was
being undertaken?
A: Officially, I don't know. I doubt it.
Q: So to your knowledge, she did not inform
her employer that she was making these measurements?
A: To my knowledge, she did not.
Q: What type of measuring device did she use?
A: Passive diffusion sampler.
Q: You're going to have to help me here
because I'm weak on this. Was this a continuous
measurement or were there –
A: No.
Q: – time samples? Could you explain what
she did.
A: The sampler relies on time integration
exposure in whatever environment you want to

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sample. And the ozone diffuses to a chemically treated filter, and then it reagents on that filter, and the derivative products are then analyzed.
Q: Okay. That's a fourth original research project that you recall. Are there any more?
A: Yes, there is one more.
Q: I'm sorry, Doctor, let me ask you another question first. Was she able to derive information from these measurements that would be relevant to a time-weighted three-hour standard?
A: Some of the measurements were exposed for a shorter duration; I believe three hours was the target time.
Q: And do you recall what results she got for those measurements?
A: No. That's part of what we have to do is sort out all the data based on the time integration and the routes and time of year.
Q: Thank you. You were about to identify for us another original research project.
A: Yes. We assisted Quantas Airlines in making similar measurements of ozone.
Q: When was that study done?
A: Approximately two years ago to a year and a

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half ago.
Q: And could you describe that study for us.
A: We essentially prepared those same samplers that we used in the anonymous airline study and --
Q: I'm sorry, did you say the "anonymous" airline study?
A: Yes.
Q: Not any longer. Go ahead.
A: Couldn't get that by you, huh? We sent those samplers to someone in Quantas who then conducted the actual exposure study.
Q: How many samples were taken?
A: I recollect something on the order of 50.
Q: I want to make sure I understand this. Is it correct that you sent the collection devices to Quantas without analyzing them yourself?
A: No, they sent them back and then we analyzed them.
Q: And what did you find; what were your results?
A: Virtually no ozone detected.
Q: On any of the samplers, no ozone?
A: I recall that these were either nondetects

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or extremely low.
Q: And are you in possession of those data?
A: No, I don't have them.
Q: Who has them?
A: Well, we sent them back to Quantas, or my engineering technician, I put him in charge of that. It was just a small-scale project.
Q: Would your engineering technician have possession of the data?
A: He might.
Q: And what's his name?
A: Thomas Dumyahn, D-u-m-y-a-h-n.
Q: Okay. Any others?
A: No.
Q: Any other original research on the airliner cabin?
A: No.
MR. FURR: Let's mark this.
(Document marked Spengler Exhibit 1 for identification)
Q: Dr. Spengler, you've been handed an exhibit which has been marked as Spengler Deposition Exhibit 1, which is a letter that we received from Mr. Rosenblatt disclosing you as an expert in a case for

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the Plaintiffs and describing the testimony that you were expected to provide in the case. Have you ever seen this letter before?
A: Yes.
Q: Did you draft it?
A: No.
Q: When did you first see it?
A: This morning.
Q: Is there anything in the letter that you disagree with with respect to your testimony, other than the first paragraph?
A: (Witness reviews document) No.
Q: You say you first saw the letter this morning. Had anyone discussed the contents of the letter with you prior to this morning?
A: Yes.
Q: And was that Mr. Rosenblatt?
A: Yes, it was.
Q: And did he call you to obtain your approval of the disclosure statement?
A: No. In my discussions of where I might be useful in this case, we went over these issues.
Q: What did you tell Mr. Rosenblatt about where you might be useful in the case?

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A: I think it was the other way around.
Q: Okay. And what did Mr. Rosenblatt tell you about where you might be useful?
A: It's in the area of being able to testify as to the levels of particulate matter, specifically tobacco-related particulate matter, that may be – probably is in some cases – that had been obtained in aircraft, commercial aircraft.
Q: Is that a subject that you're prepared to address?
A: Yes.
Q: Okay. I want to ask you a series of questions now about a report entitled The Airliner Cabin Environment, Air Quality and Safety, by the National Academy.
MR. FURR: It's my understanding that this report has been marked in a previous deposition, and there's an agreement that rather than mark additional copies in each deposition that that will be the official copy. Is that true?
MR. HOAG: That's correct. I know we had that in Dr. Stedman's –
MR. DAVIDSON: Well, no, the original report has been marked in the deposition of Paul

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Halfpenny and none of us as we sit here recall the number, but that is the master report for the purposes of all depositions taken in the case; that is, The Airliner Cabin Environment, Air Quality and Safety report.
MR. FURR: In any event, we won't be marking another copy today.
Q: Doctor, is it correct that you have a bound copy of the report in front of you?
A: Yes, I do.
Q: In the disclosure statement it says that you will "describe all facets of the study and the unanimous recommendations of the Committee that there be a ban on all smoking on domestic commercial flights." Are you prepared to address that topic?
A: The latter part I can. It's obviously some time since we met as a committee and worked on this document, so there may be some facets of the committee's deliberation and the content of this report that I don't have immediate recall of.
Q: Okay. Do you have any understanding of how the report came to be done in the first place?
A: Yes.
Q: Can you tell us what that understanding

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is.
A: That Senator Inouye requested that the FAA undertake a study specifically commissioning the National Research Council, and that study was to examine the issue of health and safety in the cabin environment of commercial airplanes.
Q: Okay. Is it correct that – actually, Congress mandated that the National Academy of Sciences conduct such a study and that the National Academy delegated it to the National Research Council?
A: That's possible. I'm not – that's possible.
Q: Doctor, the National Research Council created a new committee called the Committee on Airliner Cabin Air Quality; is that correct?
A: A new committee, specifically –
Q: To conduct this study.
A: Oh, yes.
Q: As we go forward today, if we refer to "the committee," can we agree that we are in fact referring to the National Research Council's Committee on Airliner Cabin Air Quality?
A: Yes.

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Q: Now, you were named vice-chair of that committee; is that correct?
A: Yes.
Q: How did you get involved with the committee?
A: I was called by staff people on the national – from the National Academy and asked to serve on the committee.
Q: Do you know how you came to be named as vice-chair of the committee?
A: No.
Q: What were your duties as vice-chair of the committee?
A: To assist the chair in overseeing the preparation of the document, editing the document, assisting with the committee deliberations.
Q: Were there any parts of the final report that you were not involved with?
A: I'm not sure what you're referring to. The process by which these things are done, various committee members are delegated the primary task of researching or developing the text of certain components of the report. It is brought together, it's edited by staff members, and finally then

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edited by academy staff, and the final text is submitted back to the committee for review and approval.

Q: Dr. Spengler, we've been told by other witnesses in this case, including your co-vice-chair, Dr. Halfpenny, that there was unanimous agreement on the committee with respect to all recommendations and findings of the committee embodied in the report. Is that your recollection?

A: That's my recollection. You know, if it wasn't so, the academy allows the possibility for minority opinions, and on other studies that sometimes occurs.

Q: Is it correct there were no minority opinions filed with this report?

A: That's correct.

Q: Let's turn to Pages vi and vii of the report, which is in the preface, and let's see if we can pin down what it is the committee set out to do. Beginning with the bottom paragraph on Page vi, the report states that "Issues the Committee addressed included the following: Are there problems with the air quality in commercial airlines? If so, what is the potential public

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health significance for those exposed over the short or long term? Are the problems solely those of brief discomfort, or are the health and safety of crew and passengers threatened? How well established is the threat? What can be done to alleviate it?"

Are those in fact the issues that the committee attempted to address, Doctor?

A: Well, as stated, these are the questions that we were asking about the cabin environmental health and safety, yes.

Q: Okay.

A: The specific issues I think are clear from the text. See, these aren't issues, and maybe it's semantics, but I just interpret it differently.

Q: If these aren't issues, what are they, Doctor?

A: These are questions.

Q: Well, now, when the report was written, they were obviously described as issues, so I'm not sure what the semantical issue is here, but we'll go ahead.

A: Fine.

Q: Now, the members of this committee included

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experts in various fields; is that correct?

A: Yes.

Q: Were the members of the committee collectively qualified to address those issues described at the bottom of Page vi and the top of Page vii in the preface?

A: I would have to answer that, within our limits, yes, and where we needed additional information, we sought that.

Q: In all cases in which the committee needed additional information, did it seek such information?

A: I believe within the constraints on the time and the financial resources, we did.

Q: Are there any other issues that are addressed in the report that the committee felt it really wasn't in a position to address?

A: No; if it was in this report, then I think that reflects that we were in a position to address it. The question is, can we reach resolution on all of these issues or questions stated on vi and vii? I think the point is, some more than others.

Q: In fact, on many of the application of those general issues to specific contaminants, for

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instance, the committee concluded it could not reach definitive conclusions; isn't that right?

A: On some, yes.

Q: I want to turn to the acknowledgments on Page xi. Page xi indicates that John Banzhaf, III, from Action on Smoking and Health, was one of the individuals who assisted the committee. Could you tell us how John Banzhaf assisted the committee?

A: No, I can't.

Q: Do you know who John Banzhaf is?

A: No.

Q: Are you aware that he is a lawyer?

A: No.

Q: Okay. On the next page –

A: This might help, if you're going to go through all these names. We had sort of a period where the committee sat and listened to public comments; anyone who wanted to come and say something about this issue, these series of issues, they did. We had I think a day-long session or maybe longer at the National Academy Building in Washington, and some of these names could have been people that just came and gave their position.

Q: But as you sit here, you don't know whether

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that was the limit of Mr. Banzhaf's participation or whether he participated farther; is that correct?
A: I do not recall him coming to any of our closed-session committee meetings and speaking to the committee directly, no.
Q: Did you ever speak to Mr. Banzhaf?
A: Not that I'm aware of.
Q: On the next page, Matthew Myers on the Coalition on Smoking OR Health is also listed as an individual who assisted the committee. Do you have any recollection of his role in assisting the committee?
A: No, I don't.
Q: Do you recall whether he ever spoke in the closed committee meetings?
A: It's my recollection that he didn't.
Q: Do you know who Matthew Myers is?
A: No.
Q: James Repace of the U.S. Environmental Protection Agency is also listed among those identified as having assisted the committee. Do you recall the manner in which Mr. Repace assisted the committee?
A: I don't recollect what he did.

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Q: Do you recall whether he ever spoke to closed sessions of the committee?
A: No, I don't recollect.
Q: Let's turn to talking about some of the findings of the committee in the report. Let's turn back to Page vii in the preface. In the second full paragraph on that page, the last sentence, the report states that "Cigarette smoking might be primarily a comfort issue for both nonsmokers exposed to smoke and smokers deprived of their stimulant; it might also be a health issue for nonsmokers as well as smokers; it is certainly a safety issue if cigarettes are improperly disposed."
Dr. Spengler, is it correct that the committee was unable to determine whether cigarette smoking aboard airliners was primarily a comfort issue or a health issue?
A: I have to say, the committee members expressed divergent opinions on that issue of which it was; was it one or the other or both?
Q: And so is it fair to say that the committee was able to reach no consensus about whether it was primarily a comfort or health issue?

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A: How to do fairness to all the committee members. You have to realize, this takes us back a decade here.
Q: Let me ask the question this way, Doctor. You told us if there had been a dissenting opinion from this statement, there was an opportunity for committee members to file a dissent report; is that correct?
A: Well, I think my response was to the conclusions.
Q: Actually, the question was all conclusions and findings in the report.
A: And findings.
Q: Yes, sir.
A: And the conclusions and findings – well, I think there is consensus to this statement.
Q: Okay.
A: And this statement, if you look at it carefully, sort of forges that compromise in opinions. Some people thought it was a comfort issue; some people thought it was a health issue; and everyone thought it was a safety issue.
Q: Okay. Let me ask the question this way: It is correct, isn't it, that there was no consensus

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that cigarette smoking aboard aircraft was a health issue for crew members?
A: To be clear, I think the lack of consensus, if there was indeed one, on that issue was because there was no definitive study based on health outcomes in crew members that at that time could definitively link it to their exposure to cigarette smoke. Do you see the distinction there? No one did a study on that population. But people fully agreed, and I think there was consensus – I know there was consensus on the committee – that health consequences were a likely outcome from exposures to cigarette smoke on airplanes.
Q: Can you show me a single passage in this 300-page report that makes that point, Doctor?
A: No; it's part of the deliberations of the committee as we discussed this. I think there are passages in this that admit fully that at the time of this writing – and probably it's true today, to tell you the truth – that there hasn't been any well-designed study to this occupational group of people.
Q: That addresses whether cigarette smoking aboard aircraft is linked to an increased incidence

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of disease among this population; is that correct?
A: That's correct.
Q: And that study doesn't exist even today?
A: To the best of my knowledge, it does not.
Q: And when the committee released this
report, the committee believed that such a study was
an important one; is that correct, Doctor?
A: That's correct.
Q: And without such a study, the committee
declined to reach the conclusion that cigarette
smoking aboard aircraft was primarily a health issue
for the crew; isn't that correct, Doctor?
A: We failed to reach consensus on that issue.
Q: Okay.
A: To the best of my recollection. I mean,
the other committee members might tell you something
differently, but I do recall that there were
discussions about this.
Q: Doctor, if consensus had been reached on
that issue, isn't it likely that that consensus
would be represented in this report?
A: Well, this sentence would have been worded
differently, let's put it that way.
Q: Okay. Let's turn to Page viii of the

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report, of the preface still, Doctor, the second
full paragraph on Page viii, beginning in the middle
of the paragraph.
A: This is –
Q: Still in the preface. There's language
that states, "Development of support for a complete
ban was gradual, as the evidence of contamination
and the impossibility of adequate cleansing of the
cabin air became more and more apparent. The coup
de grace to smoking in airliners was the realization
that diminished ventilation with outside air and
increased recirculation of air, a characteristic of
almost all new airliner models, will increase
previous levels of toxic products of cigarette
smoking in nonsmoking sections of the cabin."
Now, Doctor, is it correct that what the
committee is saying there is that the recommendation
for an airliner smoking ban was not based upon
concerns over past or existing ETS exposures but
speculation about what the health implications might
be for future higher exposures?
MR. HOAG: Object to the form of the
question. The sentence says exactly what it says,
and that's what the committee found.

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Q: Doctor, go ahead and answer, please.
A: Don't take offense, but your question is
absurd. I mean, if you're talking about a ban to
take place in the future, what could it possibly do
for prior exposures? They would have already had
occurred.
Q: Doctor, I said prior or existing.
A: Well, so your question was – say your
question or have it read back, if you don't mind.
Q: Okay. The committee – this is really just
a different way of expressing what we just talked
about, Doctor. And that is, the committee could not
conclude that the then existing airline cabin
exposures to ETS constituents was enough of a health
issue to recommend a complete ban; isn't that
correct, Doctor?
A: I see the point that you're making.
Q: If it's still an absurd question, tell me,
and I'll try to restate it.
A: I see what you mean. When you talked about
prior exposures, I got stuck on that. I think it
goes back to the same point I made before, that in
the absence of a definitive study or a series of
studies on this particular population, passengers

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and crew members, some committee members felt that
they could not make the prima facie argument that
health effects were occurring. Others were willing
to accept the high probability, even causality,
based on studies that had been published where
exposures had occurred in other settings.
Q: Okay, let's try it this way: Can you turn
to Pages 6 and 7 of the body of the report,
beginning with the bottom of Page 6 and turning over
to Page 7. The report states that "The committee
recommends a ban on smoking on all domestic
commercial flights for four major reasons: to
lessen irritation and discomfort to passengers and
crew, to reduce potential health hazards to cabin
crew associated with ETS, to eliminate the
possibility of fires caused by cigarettes, and to
bring the cabin air quality into line with
established standards for other closed
environments."
Now, those in fact were the bases for the
committee's recommendation, weren't they, Doctor?
A: Yes.
Q: Now, the committee describes the health
hazards to cabin crew associated with ETS as

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"potential"; is that correct?
A: That's what this says, yes.
Q: And there was, as you told us, there was a consensus on the major findings in the report; is that correct?
A: That's correct.
Q: The committee does not describe the health hazards of ETS associated with exposure in the airliner cabin as proven or anything else more definitive than "potential"; is that correct, Doctor?
A: Well, I'm repeating my answer, but I'll do it again. There's no specific study on this specific population of passengers and crew.
Q: Were you finished, or were you continuing?
A: I'm finished.
Q: Well, your answer implies that there was something less than a consensus among the committee as to whether the health implications of ETS exposure aboard aircraft were only potential. Now, haven't you told us that all major findings of this report were unanimously agreed to?
MR. HOAG: Object to the form of the question. Compound, it starts with something other

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than a question, it starts with a statement, and then it finally ends with a question.
Q: Go ahead, Doctor.
A: I think we're going around on the same point, obviously. The conclusions were unanimously accepted; we all agreed to these series of findings and recommendations as presented in the Executive Summary. And I think the use of the word "potential" health hazard is well chosen.
Q: In your opinion, that word was well chosen?
A: Well, no, in my opinion, it is well chosen for the specific context of this meeting of this committee – excuse me, of this committee – because some committee members were not as convinced as other committee members that the reduction of ETS exposure would result in concomitant health benefits; in other words, reduction of illness, et cetera, because those studies hadn't been done.
So I don't know what more we can say on this. I mean, you certainly can have consensus on the final conclusions and findings, because the way they're worded, it was deliberately structured to get an agreement among all committee members, we could agree to this.

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Q: So there was consensus that this was the proper way to express the committee's conclusions?
A: Yes. Individuals differed on the specifics, but people agreed with this general statement.
Q: Doctor, you just used a phrase that I'm going to go back to, and that is, you said that "some committee members were not as convinced as other committee members that the reduction of ETS exposure would result in concomitant health benefits." No one knows whether the reduction of ETS exposures aboard aircraft have some concomitant health benefits to flight attendants, do they, Doctor?
A: I don't know if that analysis has been done.
Q: To your knowledge, that analysis has not been done, has it?
A: To my knowledge, it has not.
MR. FURR: Let's go off the record for a minute.
(Recess)
BY MR. FURR:
Q: Doctor, let's turn to Chapter 5 of the

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committee's report. Chapter 5 would be entitled "Cabin Air Pollutants: Sources and Exposures."
A: Give me a page number, please.
Q: That begins on Page 113. But let's specifically turn to Page 131. Doctor, that's where the committee begins its report on "Environmental Tobacco Smoke"; is that correct?
A: Yes.
Q: Within the section beginning on 131, let's turn to Page 141, the subheading "Health Effects in Airplanes."
A: Yes.
Q: In that section the committee is providing this report on its findings with respect to the health effects in airplanes of ETS exposures?
A: I'm checking.
Q: Sure, take your time.
A: I want to see if there was another section that talked about it in more detail. Well, this chapter with sources and exposures. Chapter 6 was "Health Effects Associated with Exposure to Airliner Cabin Air." So we'd have to look through Chapter 6 to see if there's additional information related to the health effects.

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Q: Okay. We can do that. But is it correct that the subheading "Health Effects in Airplanes" on Page 141 refers to the committee's report on ETS exposure in airplanes?
A: Yes, with the possibility there's other ETS-related information elsewhere, but yes.
Q: In this section beginning on Page 141, the committee identifies irritation as a health effect of ETS exposure aboard airplanes; is that correct?
A: That's correct.
Q: Now, in that section, irritation is the only effect of ETS exposure aboard aircraft that the committee was able to reach a consensus on; is that correct?
A: Well, I think as it says, this is the only piece of information that we could identify after an extensive review of the literature that related specifically to health effects, definition, irritant effects that had direct relationship to airplane environment. See, I don't think there's a consensus issue here at all; this is the data. So consensus was that yes, this data exists and it applies to airplanes.
Q: In other words, Doctor, after, as you

described it, a comprehensive review of the scientific literature, the only evidence that the committee could find with respect to health effects in airplanes for ETS was irritation?
A: Yes.
Q: In the second sentence in that section, the committee states that "Irritation affects general health and welfare and thus affects performance of the crew"; is that correct, Doctor?
A: That's correct.
Q: How does irritation affect general health of the crew?
A: We're talking about irritation to the eyes, nose and throat in this case, and I think the issue here is that constant and persistent irritation of that nature could aggravate preexisting conditions, make them worse, or chronic exposures to these conditions could precipitate other health response of the respiratory tract and sinuses.
Q: Doctor, there are many sources of irritation to the eyes, nose and throat of airline crew members, aren't there?
A: I didn't quite follow the question.
Q: There are many causes, other than

environmental tobacco smoke, which are a source of irritation to the eyes, nose and throat?
A: In life? In general?
Q: In the airliner cabin.
A: Not really.
Q: Well, isn't low humidity a source of irritation to the eyes, nose and throat?
A: No, it's not an irritant, no, it wouldn't be. It may change your responsiveness to irritants; it might dry out the mucus-secreting regions of the ear, nose and throat, but in itself wouldn't be an irritant.
Q: Is ozone an irritant?
A: Yes, ozone is an irritant.
Q: What other irritants do the crew members encounter in the airliner cabin?
A: Specifically to the airway, right?
Q: Yes, sir.
A: It could be transient emissions of irritating gases and vapors.
Q: What gases or vapors do you have in mind?
A: Those that might be related to engine exhaust during ground operations; those that may be related to passengers' applications of nail polish

remover, for example.
Q: Are VOCs an irritant?
A: They would be included in those class, but not all VOCs are irritants. Some specific compounds are, but not all would be considered irritants.
Q: Are particulates an irritant?
A: By that you mean just general dust?
Q: The particulates in the airline cabin.
A: I would say under the circumstances in the absence of smoking, no, they're not.
Q: Turn to Page 141 of the report, Doctor.
A: We're on that page, yes.
Q: Okay. Just give me a moment. I don't see what I was going to ask you so we'll try something else.
Let's go back to the process for just a moment. Is it correct that each committee member generally had responsibility for various chapters in the report?
A: Not various chapters but sections of various chapters. In other words, they may have been responsible for sections that appeared in different chapters; it wasn't subdivided chapter by chapter.

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Q: What sections did you have specific responsibility for?
A: I drafted part, if not all, of this section that we're just talking about now on the sources and concentrations of ETS, and I drafted a section on cosmic radiation.
Q: Okay.
A: And I contributed to drafting various other sections in my responsibility as vice-chair. I can't recall specifically which pieces they were.
Q: Let's turn to Page 143, Doctor, the top of the page. I found what I was looking for. The first sentence of that paragraph reads that "The possible health effects of ETS on nonsmokers cannot simply be extrapolated from the health experience of active smokers, for the following reasons."
Now, you agree with that principle, don't you, Doctor?
A: Yes.
Q: And there is a consensus on the committee that that was the case?
A: Yes.
Q: Let's turn to Page 146 of the report. Let's go to the bottom of the page, the last three

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sentences. I believe the report states, "There were no assessments of ambient conditions or biochemical measures of components of tobacco smoke in most studies. Therefore, it is difficult to extrapolate the results of these studies to potential health effects in passengers and crew members in airliner cabins."
You agree with that conclusion, don't you, Doctor?
A: Let me just check what is referred to by "those studies."
Q: Sure.
A: I'm not familiar with every study cited here, or at least it's been a long time since I've read them, but I will assume that when we wrote this statement that that certainly is accurately depicting the situation with reference to those studies.
Q: Okay. Dr. Spengler, have you reviewed or even seen the complaint filed by the Plaintiffs in this case?
A: Yes, I did.
Q: Have you seen the list of 23 diseases that the Plaintiffs allege in their complaint as being

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experienced by the class due to their ETS exposures?
A: Like I said, I read the complaint, but I could not recite all those diseases.
Q: Doctor, we've been talking about what the committee concluded with respect to ETS exposure aboard airlines. Let's tie this up by reference to the allegations in the Plaintiff's complaint, and we can move on to a new section.
Doctor, isn't it correct that the committee reached no conclusion with respect to whether the ETS exposures aboard airliner cabins caused or was causing cancer of the lung in crew members?
A: I think –
Q: Let me rephrase that question.
A: Please.
Q: Doctor, the committee evaluated the available information with respect to the health effects of ETS exposure aboard airliners; is that correct?
A: Yes.
Q: I'll make a series of statements, and you can just tell me whether that's correct or not. Okay?
A: All right.

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Q: The committee did not conclude that ETS exposure in airliner cabins causes cancer of the lung in nonsmoking crew members?
A: That was not a specific finding or conclusion, that's correct.
Q: The same would be true for cancer of the larynx in nonsmoking crew members?
A: That's correct.
Q: And the same would be true for cancer of the oral cavity in nonsmoking crew members?
A: That's correct.
Q: And the same would be true for chronic bronchitis in nonsmoking crew members?
A: Correct.
Q: The same would be true for emphysema in nonsmoking crew members; is that correct?
A: Correct.
Q: And the same would be true for coronary heart disease or cardiovascular disease in nonsmoking crew members?
A: That's correct.
Q: The same would be true for cancer of the esophagus in nonsmoking crew members?
A: Correct.

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Q: The same would be true for cancer of the bladder in nonsmoking crew members?
A: That's correct.
Q: The same would be true for cancer of the kidney in nonsmoking crew members?
A: Correct.
Q: The same would be true for cancer of the pancreas in nonsmoking crew members?
A: Correct.
Q: The same would be true for cancer of the stomach, cervix or breast in nonsmoking crew members?
A: Correct.
Q: The same would be true for endometritis in nonsmoking crew members?
A: Correct.
Q: The same would be true for cerebrovascular disease in nonsmoking crew members?
A: Correct.
Q: The same would be true for intrauterine growth retardation in nonsmoking crew members?
A: I don't recall that being in the findings of the committee.
Q: The committee made no findings on

conclusions regarding the ETS exposures aboard airliners on infertility of nonsmoking crew members?
A: That is correct.
Q: The same would be true for other complications in pregnancy?
A: Specifically for ETS?
Q: Yes, sir.
A: I think that's correct.
Q: The same would be true for infant mortality?
A: Correct.
Q: Peptic ulcer disease?
A: Correct.
Q: Aggravation of asthmatic conditions or allergies?
A: Other than the citation we made about irritation effects, and I would say that that's included within the constellation of diseases that would be aggravated by irritation effects.
Q: Now, for all those diseases that I just listed, the committee found no evidence that allowed it to conclude that ETS exposure aboard airliner cabins was causing those diseases in nonsmoking crew members, is that correct, except for the asthmatic

issue which you raised?
A: Well, you sort of put that last thing, no evidence –
Q: No evidence that would allow it to conclude.
A: Well, no direct health evidence, but evidence from concentrations. So that's part of the evidence.
Q: Okay.
A: And from that evidence we concluded that it was possible that there were health consequences as a result of exposures that were occurring at that time. So there's a fine distinction I see in my mind. I know – and just so we don't go on to a whole series of questions about this, I'll repeat – that at the time of this publication, and I think it still is true today as we talk, that there's no definitive occupational health study that can substantiate these effects occurring differentially in the nonsmoking flight crew as a result of their exposures to cigarette smoking. It doesn't exist in the literature.
Q: But the committee also found no other type of evidence that it was comfortable relying upon to

conclude that the airliner ETS exposure was a proven or definitive cause of any of those diseases that we've just talked about in nonsmoking flight attendants, did it?
MR. HOAG: Object to the form of the question.
A: I mean, they did – we did rely on concentration data, and we did rely on published data in other populations and said that it was reasonable to conclude that this was analogous by way of exposures to those studies that did show effects. Now, so the fine point is "definitive." I mean, the answer is no, to answer your question directly, there was no definitive evidence. But you saw the conclusions, we went over them, that the findings are quite clear that in... I'll just end the answer there.
MR. FURR: Let's grab a sandwich.
(Whereupon, at 1:15 p.m. the deposition was adjourned for luncheon recess)

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AFTERNOON SESSION (1:30 p.m.)
BY MR. FURR:
Q: Dr. Spengler, let's turn to discussing some of the other airliner cabin contaminants that were addressed by the committee in the '86 report. Let's begin by discussing the ozone. Could you explain to us what ozone is, please.
A: Yes. Ozone is a molecule that's comprised of three oxygen atoms.
Q: And what's the source of ozone in the airliner cabin?
A: The source is outdoor air, ozone in the outdoor air.
Q: How is ozone formed?
A: It's formed as a chemical reaction between - well, back up. To be specific for airplanes, the concern is for stratospheric ozone, and that is formed by the direct dissociation of the oxygen molecule by ultraviolet radiation and that temporary recombination to form oxygen triplet molecules.
Q: Doctor, could you turn to Page 113 of the committee's report, please. Right under the heading "Ozone in Commercial Aircraft Cabins," the first

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sentence reads, "Ozone is present in the atmosphere as a consequence of the photochemical conversion of oxygen by solar ultraviolet radiation." Is that the process that you were just describing, Doctor?
A: Yes.
Q: The next sentence reads that "A marked and progressive increase in ozone concentration occurs between the tropopause and the stratosphere; i.e., it occurs within the flight altitude of commercial aircraft." Is that correct, Doctor?
A: Yes.
Q: What are the health consequences of ozone exposure?
A: The consequences that have been noted to date include direct irritant effects that lead to pain upon breathing, further airway irritation that might aggravate, again, preexisting respiratory diseases. It has also been associated with decrease in pulmonary function on a temporary basis. Very recently there have been the implications that the coexposures of ozone and allergens tend to enhance the effects of those allergens. Ozone has also been associated with increases in asthmatic symptoms - symptoms of asthma in people that have preexisting

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asthma.
Q: Doctor, is there an issue with respect to the health implications of exposure to ozone and radiation together?
A: "Radiation" is a really broad definition.
Q: Okay.
A: Because there's ultraviolet radiation and there's ionizing radiation and lots of other kinds of radiation.
Q: Would you qualify that, the type of radiation experienced by flight attendants in the airliner cabin.
A: Well, again, it will sound like I'm argumentative here, but there's lots of radiation. There's electromagnetic radio frequencies. There's a whole range of radiation that they might be exposed to.
Q: Let me restrict the question then to ionizing radiation.
A: All right. So the question is, is there a concern for the coexposures to ozone and ionizing radiation?
Q: Yes.
A: I wouldn't think so, no.

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Q: Are you familiar with any literature on that subject?
A: No.
Q: Could you turn to Page 116 of your report. At the bottom of the paragraph, the partial paragraph, the first paragraph on the page, the report states that -
A: I'm not with you yet.
Q: Sure.
A: The bottom of the first paragraph?
Q: The paragraph beginning at the top of the page, which is a paragraph addressing health implications of ozone exposure, which you identified all of them as far as I can tell. The last sentence reads that "If exposure is repeated within 24 to 48 hours, pulmonary function decrements are markedly greater." Do you agree with that statement, Doctor?
A: I'm going to see if I recognize that particular citation. It's done by Steve Horvath, and I agree with that statement.
Q: Now, repeated exposures within 24- to 48-hour periods are the types of exposures that flight attendants experience, aren't they?
A: Possibly.

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Q: Let's turn to talking about radiation, Doctor. I believe you told us that you had responsibility for the radiation section of the committee's report; is that correct?
A: To the extent that I drafted the first or wrote the first draft, yes.
Q: You began listing these earlier. What types of radiation are the crews of commercial airliners exposed to?
A: Well, there are several ways you can classify radiation. I think just for completeness there are two categories: one is ionizing and the other is nonionizing radiation; and within the realm of nonionizing radiation it's the full spectrum of the electromagnetic wavelengths, including microwaves and radio waves.
And so to some extent the flight attendants might be exposed to various radio frequencies because of the electronic equipment on board. I don't believe they use microwaves to heat food, but that would be another source if they did. But put all that aside, I think the committee recognized and focused on the potential for cosmic radiation to be a concern.

Q: Now, cosmic radiation is the ionizing radiation; is that correct, Doctor?
A: Yes, it is.
Q: And what are the sources of ionizing radiation that the crew of an airliner are exposed to?
A: Well, primarily if it's in the form of cosmic radiation, it would be essentially that high energy radiation in the form of small energetic alpha particles and protons that cascade through our atmosphere from solar and galactic sources.
Q: Is there a relationship between the altitude that an airplane flies and the dose of ionizing radiation that the crew receives?
A: I'd rephrase it slightly.
Q: How would you say it, Doctor?
A: There's a relationship between the altitude that the plane flies and the concentration that they receive. There are other variables.
Q: What is that relationship?
A: The higher the altitude, the less depth of atmosphere above them that would provide some shielding from cosmic radiation.
Q: Okay. So airliner cabin crew members are

exposed to a higher concentration of ionizing radiation in the airline cabin than we are on the ground; is that correct?
A: That's correct.
Q: Is there also a relationship between the latitude that a plane is flying at and the concentration of ionizing radiation that the crew members are exposed to?
A: Yes, there is.
Q: And what is that relationship?
A: The further away the flight path is from the equator, and that would be either pole, there is a higher concentration of radiation. But it's the same phenomenon - well, back it up, there are two phenomena. But it's the same in principle as that, the depth of the atmosphere is actually thinner over the poles than it is over the equator. So indirectly by being at a higher latitude on the surface of the Earth at a given altitude, you also have less atmosphere above you.
Q: Dr. Spengler, is it correct that the chief health risk that the crews of commercial airliners face from ionizing radiation are neoplastic or carcinogenic diseases and reproductive illnesses?

A: I have trouble lumping them all together. It's not reproductive illnesses. It might be malformations; it might be an unwanted reproductive outcome.
Q: I should ask the question this way: What are the health risks that commercial airline crews face from ionizing radiation exposure in the airliner cabin?
A: They would be those risks in which damage to genetic material would be the precipitating event. So they include a whole variety of potential soft tissue tumors, and including reproductive outcomes if the origin of those outcomes are from damage to stem cells.
Q: Are you finished?
A: Yes.
Q: Are you familiar with the types of tissue that are most sensitive to developing cancer from exposure to ionizing radiation in humans?
A: No.
Q: Let's go back to the committee's report, Doctor. And you raised this topic earlier, but let's talk about the committee's review of airliner crew members' exposure to ground fumes. I think

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that we can turn to Page 130 of the committee's report. At the bottom of Page 130, the top of Page 131, the report states that "Aircraft jet engines emit a variety of potentially toxic substances, including carbon monoxide, oxides of nitrogen, hydrocarbons, aldehydes (especially formaldehyde), particles, and polynuclear aromatic compounds"; is that correct, Doctor?
A: Yes.
Q: What are the health implications of those exposures, Doctor, to ground fumes?
A: It could be a variety of health implications, from irritation of the respiratory tract, to increased risk of cancer, depending on all the conditional variables: dose, duration, all the other concentration.
Q: Is it correct in an aircraft, jet engines emit substances that we think of as carcinogens?
A: Similar to all combustion engines, yes.
Q: And the crew of a commercial aircraft is a population that is one of the most exposed populations to the emissions from jet engines; is that correct?
A: I doubt it. Probably the ground crew would

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be.
Q: Are there any studies of the health risk from exposure to ground fumes in a commercial aircraft crew?
A: None that I know of.
Q: The ground fumes also themselves, many of the components of the ground fumes would be irritants, wouldn't they, Doctor?
A: Carbon monoxide wouldn't be. Aldehydes certainly could be, some of the hydrocarbons. Particulates could be if in high enough concentration.
Q: And so those are all irritants that the crew of a commercial aircraft would be exposed to in the airliner cabin; is that correct?
A: They could be exposed. That's not – obviously it's not a condition they encounter on all occasions.
Q: Let's talk about relative humidity aboard the airliner cabin, Doctor. What does the term "relative humidity" mean?
A: It refers to a ratio expressed as a percent of the amount of water vapor that is in the air to the amount of water vapor that could possibly be in

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the air if that air were saturated.
Q: Is relative humidity an issue in the airliner cabin?
A: Yes.
Q: Can you tell us what you know about that, please.
A: I know that the relative humidities in aircraft represent conditions that are very dry, equivalent to – strike that – ranging from 5 percent to 25 percent relative humidities during the cruise portion of the flight.
Q: And what are the comfort or health consequences of being exposed to relative humidities in that range?
A: The health consequences are undocumented. After all, there are many populations in the world that live in such dry conditions. The comfort implications are those that I referred to earlier, and they include the drying out of the moist regions of the nose, throat and eyes, leading to potential irritation from other substances. There is the possibility – it's been referred to in some of the literature – that transboundary migration of materials may be increased under very dry

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mucus-secreting region conditions. I think people with contact lenses under these dry conditions also experience more eye irritation, and under prolonged flights there may be more complaints about dry skin.
Q: Do you have an opinion as to whether exposure to low relative humidity affects lung capacity?
A: I know of no evidence that would support that.
Q: What about whether exposure to low relative humidities affects the risk of respiratory infections?
A: I know that that has been suggested by flight attendants and maybe even passengers; but, again, there's no factual evidence of that.
Q: Could you turn to Page 215 of the committee's report, please. In a section entitled "Desirability and Feasibility of Additional Data Collection" and a subsection "General Concepts and Approaches," top of the second paragraph on Page 215, the committee stated, "Simply measuring the contaminants and other relevant variables of the airliner cabin does not address the question of the likely health effects of short or long exposure to

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that environment. The evaluation of the health
effects of exposure requires the collection and
interpretation of data very different from those on
exposure."
Do you agree with that statement, Doctor?
A: Yes.
Q: What type of data are being referred to
there?
A: I believe what we were referring to would
be the collection of data on symptoms, data on
alterations in function, data on frequency of
disease within these exposed and nonexposed
populations.
Q: Let's apply the embodied language we're
looking at, environmental tobacco smoke. Would you
agree, then, that simply measuring the environmental
tobacco smoke aboard the airliner cabin does not
address the question of the likely health effects of
short- or long-term exposure to that environment?
A: No, I don't think it would, no.
Q: Why not? Strike that. My question is, why
is ETS different than anything else in this regard?
A: Well, I wouldn't say that either. I'd say
I could think of some pollutants; ozone, ETS, even

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the effects of ionizing radiation, that we could
learn a lot about the likely health effects from
long- or short-term exposures by doing carefully
constructed experiments in other settings. And then
by understanding the concentrations experienced in
the aircraft environment, we could say a lot about
the likelihood of health effects.
Q: Okay.
A: As an example, a lot of our evidence on the
health effects of ozone has come from controlled
chamber studies.
Q: Is what you're telling me, Doctor, that if
you can establish a dose-response relationship for a
contaminant and health end point, you may be able to
in some environment – then you may be able to take
that dose-response relationship and apply it to the
concentration of a contaminant that's found in the
airliner cabin environment?
A: That's correct.
MR. FURR: Let's mark this.
(Document marked Spengler Exhibit 2
for identification)
Q: Dr. Spengler, you've been handed an exhibit
that's been marked as Spengler Deposition Exhibit 2,

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which is a reprint of an article entitled "Health
Effects and Sources of Indoor Air Pollution, Part
I." There's a John D. Spengler listed as one of the
three authors of that article. Is that you?
A: Yes.
Q: Now, this article was published in 1987; is
that correct?
A: Yes.
Q: So it was published after the NRC report on
airliner cabin air quality?
A: Yes.
Q: And it was published after the 1986 Surgeon
General's Report; is that correct?
A: Yes.
Q: Now, in the upper left hand of the first
page, the words "State of Art" appear. What do
those words signify, Doctor?
A: This refers to a section of this journal
that is set aside for reviews of any number of a
variety of topics. It is not part of our title.
Q: I take it, then, that this is a section of
the journal reserved for comprehensive reviews of
issues that are meant to bring the available
information up to date for the reader?

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A: Yes.
Q: One of the indoor pollutants that you and
your coauthors reviewed was environmental tobacco
smoke; is that correct?
A: Correct.
Q: Could you turn to Page 1490 of that
report.
Let me ask you this general question
first. Now, in your review of environmental tobacco
smoke in this article, you and your coauthors were
reviewing the information available with respect to
environmental tobacco smoke or ETS exposure and
health impacts in general and not as restricted to
the airliner cabin; is that correct?
A: That's correct.
Q: In the middle of Page 1490 there's a
heading "Tobacco Smoke." Let's take a look at the
last section in the first paragraph under that
heading. That reads, "Consequently, the evidence on
involuntary smoking is more limited in scope than
for active smoking, and controversy remains
concerning certain associations of involuntary
smoking with disease." Do you see that, Doctor?
A: Yes.

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Q: What certain associations were you referring to there?

A: I think at the time of this publication we were referring to associations between passive or involuntary smoke and cardiovascular disease.

Q: Let's turn to Page 1492 of your report. On the far right-hand column there's a heading of "Nonmalignant Respiratory Effects." And in that column you review nonmalignant respiratory effects in children, I believe, to ETS exposure, but then you finish the paragraph with the sentence, "Only limited data pertaining to adults have been available, and definitive conclusions have not been made for adult populations"; is that correct?

A: That's correct.

Q: Now, the nonmalignant respiratory effects that that sentence applies to would include emphysema and chronic bronchitis, wouldn't it?

A: Let me quickly review the paragraph.

Q: Sure.

A: (Witness reviews document) Your question was that this statement pertaining to adults and the limited data available includes bronchitis and what else did you say?

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Q: Emphysema.

A: Emphysema. I believe that's true.

Q: Let's turn to Page 1493. In the middle column of 1493, the second full paragraph, the first sentence reads that "The findings of the newer studies are inconsistent on the relationship between passive smoking and wheezing and asthma"; is that correct, Doctor?

A: Yes, that's correct.

Q: Now, that sentence, is it correct that that sentence is referring to the newer studies on childhood exposures to ETS and wheezing and asthma?

A: The first reference is. It's a cohort of children. (Witness reviews document)

Yes, it does refer to several studies, some that did show effects, and then at the beginning of the next paragraph it talks about a study that did not find associations with ETS to symptoms in children.

Q: Okay. Now, as a general proposition, children are more sensitive than adults with respect to the onset of wheezing and asthma, are they, Doctor?

A: I'm not sure how I understand what you

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implied. You said to the onset of –

Q: To having asthma initiated by a given exposure.

A: Well, asthma can develop in children and in adults without – well, it can develop in both. I would imagine the onset of new asthma, the rates are higher in children than in the adult population. So I believe your answer – the answer to your question is yes.

Q: Let's turn to 1494. On the far right column of 1494 you and your coauthors began your review of ETS exposure and lung cancer; is that correct?

A: Yes.

Q: Under that heading, let's go to the second paragraph, which reads, "Time trends of lung cancer mortality in nonsmokers have been examined with the rationale that temporally increasing exposure to environmental tobacco smoke should be paralleled by increasing mortality rates."

Let me ask you first, Doctor, could you break that down for us and tell us what it is that you and your coauthors were attempting to look at.

A: Well, this is the opening sentence to a

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paragraph, and it is setting up the discussion for the study that follows – two studies that follow. The hypothesis is the following: that the trends of tobacco smoking, tobacco use in the United States increased dramatically since World War II, and if that were the case, then lung cancer in those that were not direct smokers would also show an increase, because it would stand to reason that their passive exposure to a larger segment of the population would also be increased. So the point is that somebody in these two references here tried to examine whether that relationship led to an observable increase in lung cancer in nonsmokers.

Q: Would you agree, Doctor, that this is an important line of evidence to assess in attempting to determine whether environmental tobacco smoke is causing lung cancer in nonsmokers?

A: I can't recall. It might be important, but it might also be intractable that you cannot separate out all the confounding variables that lead you to conclusively define a trend. Many other things were happening too in terms of changing of substances and radon and, who knows, lots of other things.

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Q: Would you agree, Doctor, that if you could
isolate this question that this question would go
right to the heart of whether increases in ETS
exposure have been accompanied by increases in lung
cancer mortality in nonsmokers?
A: Like I said, it's one line of inquiry that
is worth pursuing, yes.
Q: And you and your coauthors pursued it?
A: No, we did not pursue it.
Q: You did not?
A: No, we described – this is a review.
Q: Right.
A: So we described the studies that examined
the trends analysis. We didn't do –
Q: I understand it's a review. You and your
coauthors examined what evidence was available on
this question?
A: Correct.
Q: And apparently you found two studies; is
that correct?
A: That's correct.
Q: And in one study there did appear to be a
parallel; is that correct?
A: Let me see what we said on that.

Q: Sure.
A: (Witness reviews document) That's correct.
Q: But you and your coauthors concluded that
occupational environmental exposure to others than
ETS might be the cause of the apparent mortality
rate increase in males, correct?
A: This could have been taken from the
discussions by the authors themselves. Without
reference back to the study, I cannot say whether
this was our conclusion or this was really the
observation limitations of the study that were
pointed out by the authors themselves.
Q: Well, let me ask you this, then. I
understand that this is a review paper. Are the
statements contained in this paper the conclusions
of yourself and your coinvestigators as to what the
studies show, or are they merely a report on what
other investigators claim their studies to show?
A: Well, it could be both.
Q: It could be both..
A: Because, I mean, we obviously have a broad
range of experiences, both from work we've done, me
and my coauthors, in this case and reading a lot of
the literature. For example, on this one, it is

possible that the authors admitted that they were
unable to control for the trends in environmental
exposures and other possible contributors, or it
could have been that when we read the paper, they
may not have said it, but we said, "Look, unless you
have controls for these other factors, you can't
determine conclusively that this trend can really be
attributed to an increase in exposure to ETS."
Q: I made a mistake a minute ago; I said that
you and your coauthors evaluated two studies. Now
having looked at this, you actually looked at three
studies, didn't you?
A: There's a reference to this Dorn study,
yes. But I think it was two. Let me just see.
Q: There's Enstrom?
A: Enstrom, and then there was Garfinkel.
Q: Who did two studies, correct?
A: Correct.
Q: And for the latter two studies, Garfinkel
at least reported no trends in the incidence of
smoking and lung cancer among nonsmokers; is that
correct?
A: That's correct.
Q: And so is it fair to say that you and your

coauthors found no support for the hypothesis that
ETS exposure causes lung cancer from this line of
evidence?
A: No; we found some support for that
hypothesis, and Garfinkel's could not support. But
you concluded we found no support, and I think it
stands for what it is. It says the evidence is
ambiguous.
Q: Okay, far enough. The evidence was
ambiguous.
Let's turn to Page 1497. Is it correct,
Doctor, that we are still in the "Lung Cancer"
section of your review of ETS?
A: Yes, I believe that is correct. Well, it
changes on this page to "Cardiovascular Disease."
Q: Let's restrict our attention to the
left-hand column, the top of it above the "Other
Cancers" for now. At the top of the page you and
your coauthors state – you and your coauthors
recognize that the Surgeon General in '86
"characterized involuntary smoking as a cause of
cancer in nonsmokers"; is that correct?
A: Yes.
Q: But then following that you state, "The

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extent of the lung cancer hazard associated with
involuntary smoking in the United States remains
uncertain, however. The epidemiologic studies
provide varying and imprecise measures of risk, and
dosimetric aspects of involuntary smoking in the
respiratory tract are not yet well understood"; is
that correct?
A: Yes, that's what it says.
Q: Well, now, by varying risk you and your
coauthors meant some of the studies showed an
increase in risk and some of the studies didn't show
an increase in risk; isn't that correct?
A: I think that you used the words both
"increase" and "increasing," and there is a
distinction. Some studies showed that the lung
cancer risk was increased in those nonsmoking women
who have spouses who smoked, and still other studies
showed that that risk increased in proportion to a
surrogate measure for dose.
Q: I'm sorry, I don't understand. Could you
explain it to me. Let me ask another question and
get us started right again here.
When you and your coauthors wrote that "The
epidemiologic studies provide varying measures of

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risk," what did you mean?
A: It means that they did not give these same
number in terms of the increased risk with a uniform
exposure. Now, it's poorly stated, let me try this
again.
Maybe it's best to give it as an example.
And I'm not citing a particular study. But let's
say that a study showed that an exposure for 20
years to a pack-a-day smoker to a person who didn't
smoke led to a 1.3 increase in their risk of lung
cancer over a baseline of 1, so a 30 percent
increase. Some studies showed that that same
exposure measure, one year – excuse me – 20 years,
one pack a day, would not show any risk increase or
some would show higher than 1.3. This is what we
meant by that, that there were inconsistencies in
the magnitude of the risk attributed to the
exposure.
Q: And in fact, some of the studies showed no
increase in risk; is that correct?
A: That's correct.
Q: And so the studies were inconsistent with
respect to whether they found an increase in risk or
not?

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A: Restating that, yes.
Q: What did you and your coauthors mean when
you said that "dosimetric aspects of involuntary
smoking in the respiratory tract are not yet well
understood"?
A: This had to do with what – that most of
these studies, in fact, I think at this time all of
these studies, relied on indirect measures of
exposure to ETS, namely, calculations based on
smoking habits, location and duration; that there is
not quantification as to how much, let's say,
particulate dose or any of the substances actually
get to the target tissue within the lungs. That's
what "dosimetry" means.
Q: Doctor, wouldn't you agree that today there
is still no quantification as to how much of the
particulate dose or any of the substances actually
get to the target tissue within the lungs?
A: I think it's better understood today.
There's better quantification of composition,
characterization of what is in the ETS; there's
better characterization of the particle size
distribution and what phases are gas phases or
condensed phases; and there are a lot more

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measurements of concentrations in a variety of
locations. So I think we are better off today than
we were at the time we wrote this in understanding
elements of the dosimetry.
Q: I'm still confused. What more do we know
today than 1987 regarding how much of the
particulate dose or any of the other substances get
to the target tissue within the lungs?
A: Well, I just recited what I feel have been
contributions to our understanding that have been
made in the intervening ten years. And in its
total, this information, including, I would have to
say, information on DNA adducts of smokers and
nonsmokers and cotinine measurements in nonsmokers,
provide a lot of information about how much the
nonsmoker is receiving via inhalation. So I can't
say that we know 50 percent more or 75 percent more;
it can't be measured that way.
Q: Doctor, as you sit here, can you describe a
dose-response relationship for ETS exposure and lung
cancer risk in nonsmokers?
A: Let's see. I think the best we can do now
is to describe an exposure-response relationship and
from there deduce the potency for a microgram per

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cubic meter ETS mixture.
Q: Okay. Can you describe an exposure-response relationship for me. Let me ask you a different way.
Would you agree, Doctor, that there is no consensus in the scientific or medical literature regarding an exposure-response relationship for ETS exposure and lung cancer risk?
A: "Consensus" is all-encompassing. There will be people that would disagree. But to the extent that you look for these three primary documents, the Surgeon General's report, the National Academy of Sciences and the EPA document on passive cigarette smoke, as a consensus among the scientific community well versed in this field, there would be an agreement that increased exposures to environmental tobacco smoke increases the probability of lung cancer in a linear fashion.
Now, if you were to say, what is the slope of that line, you might find some divergence of opinion of how steep that slope is. Do you follow me?
Q: I do, I follow what you're saying. I don't necessarily agree with it. But here's my question:

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Are you telling me that any ETS exposure, however slight, engenders an increased risk of developing lung cancer?
A: Do I personally believe that?
Q: Yes, sir.
A: No, I don't.
Q: Thank you. Do you know how much ETS exposure is required to create an increase in lung cancer risk?
A: I could make an approximation.
Q: And what would that be?
A: I would suspect that given the right population size and study design that one could detect an increased risk of lung cancer to the nonsmoking person for the equivalent of 20 years' exposure to half a pack a day in a residential setting. Now, how do you quantify that? One has to go back and calculate what that might mean by way of dose. But it's not a transient, one-time exposure, so there's...
What I said is that - what I mean by that is that you would be able to - probably at that level, you would be able to statistically see it over the noise of chance.

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Q: So at that level you believe that you might produce a statistically significant increase in risk?
A: You could see it with a properly designed epidemiologic study.
Q: Well, now, I asked you to do this - I'm not criticizing, I just want to make sure the record is clear. At this point we are in the realm of speculating, aren't we?
A: Absolutely.
Q: Now, is it correct, Doctor, that, on average - I understand it would depend on many variables, but on average - that smoking half a pack a day in a residential setting would produce a RSP level of approximately 20 to 30 micrograms per cubic meter?
A: Probably a little less than that, actually.
Q: Let's go back to your 1987 article. Staying on Page 1497, let's go to the next topic that you address, which is "Other Cancers." And go to the summary paragraph at the end of that section that reads, "These associations of involuntary smoking with a cancer at diverse nonrespiratory sites cannot be readily supported with arguments for

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biologic plausibility. Increased risks at some of the sites, e.g., cancer of the nasal sinus and female breast cancer, have not been observed in active smokers." Let me stop here and ask you a question.
Now, this was written in 1987, correct?
A: Correct.
Q: You would agree that those two cancers still are not being reported in active smokers today, wouldn't you?
A: I don't know.
Q: You don't know. The quote continues, "In fact, the International Agency for Research on Cancer has concluded that effects would not be produced in passive smokers that would not be produced to a larger extent in active smokers." This was your conclusion in 1987, correct, Doctor?
A: Correct.
Q: And this would still be your conclusion in 1997, wouldn't it?
A: Well, let's say this is the conclusion of IARC, which is the International Agency for Research on Cancer, and I would agree with this statement, yes.

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Q: If you don't see an increase in cancer among active smokers, it makes no biologic sense that you would see an increase in cancer among ETS-exposed nonsmokers; is that the point?
A: No, I guess I probably wouldn't agree with that. No, I wouldn't.
Q: Why is that?
A: For a variety of reasons. One might have – there are all sorts of physiological changes that occur in active smokers that might change the metabolic pathways of certain materials, which then in turn would change the rate at which they could apply or accumulate in fat tissue. And a passive smoker may not have that, certainly wouldn't have the physiological changes that active smokers have, and they might represent a genetically more susceptible group that have chosen not to smoke because of some tolerance level issue. So their phenotype or their genetic typing could constitute a different group.
Q: Doctor, you believe there may be a genetic component to the determination that people make as to whether to smoke or not?
A: I think I said it the other way around.

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Q: You said "they might represent a genetically more susceptible group that have chosen not to smoke because of some tolerance level issue."
A: That's right.
Q: What does that mean?
A: Well, for example, in a population of people with asthma, the percent that are smokers, active smokers, is lower than the percent in the rest of the U.S. population, for example. So by virtue of their condition, asthma, they're more sensitive to irritants and are less likely to be in the smoking population. And there is a genetic component to susceptibility for asthma. So just using that one example alone –
Q: I understand. Let's go back to your paper. So, Doctor, is it correct, in 1987, you and your coauthors concluded that there is no proof that ETS exposure was causing cancer in nonrespiratory sites in nonsmokers?
A: We cite some studies that did show those increased risks.
Q: Right.
A: We were at the point of saying that we had difficulties in making the same arguments of

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biological plausibility for increased nonrespiratory cancers, because those other tissues are not necessarily in the direct target pathways as are the respiratory tissues. So at the time that we wrote this, it is as we had concluded, the evidence was certainly not as strong for these other cancers as it was for respiratory cancers.
Q: Doctor, if you were writing this paper today, you would reach the same conclusion, wouldn't you?
A: I'm not sure. I'd have to – I don't think I'm in a position right now to answer that.
Q: Let's go to the next topic in the paper, "Cardiovascular Disease." Is it fair to say, Doctor, that you and your coauthors in 1987 found the evidence insufficient to allow you to reach a conclusion as to whether ETS exposure was a cause of cardiovascular disease in nonsmokers?
A: At the time of this writing, yes.
Q: Now let's go to the next section which is "Summary" and look at the last sentence, which reads, "In adults, involuntary smoking is a cause of lung cancer, but associations with other diseases have not been unequivocally established." That was

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your conclusion in 1987; is that correct, Doctor?
A: Yes.
Q: Doctor, you and Dr. Samet, who was one of your coauthors for the '87 paper, were also the editors of a book titled Indoor Air: A Health Perspective in 1991; is that correct?
A: That's correct.
Q: And in Chapter 6 of that book an article very similar to your 1987 article appears; is that correct?
A: That's correct.
Q: Now, you weren't actually an author of that chapter, were you?
A: No, I wasn't.
Q: Dr. Samet was?
A: If Chapter 6 was on the health effects of ETS, then he was the author.
Q: Did you review that chapter?
A: I read the whole book in editing it, but, again, that was early '90s.
Q: Is it fair to say that Dr. Samet's conclusions in 1991 were almost identical to the conclusions that you all reached in your 1987 paper?
A: It's likely. I couldn't say that without

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going back and looking at his chapter; it wouldn't be fair to him.
Q: Now, you would have reviewed that chapter in 1991, you told us; is that correct?
A: That's correct.
Q: And if you had had any disagreement with the conclusions Dr. Samet was making in 1991, would you have expressed those disagreements to him?
A: Yes, I would have.
Q: Do you recall expressing any disagreements with Dr. Samet about the conclusions that he was reaching in 1991?
A: Not that I recall. I hope you bought the book.
Q: I'm sure we did.
A: I hate to have my copyrights infringed.
Q: There are certain exceptions to the copyright laws.
A: I'm sure you'd know them.
MR. FURR: Let's mark this.
(Document marked Spengler Exhibit 3 for identification)
Q: Doctor, you've just been handed an exhibit marked as Spengler Deposition Exhibit 3. Is this

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the Chapter 6 of the book that you and Dr. Samet edited in 1991 that we just referred to?
A: (Witness reviews document) Yes.
Q: Could you turn to Page 132, please, of that chapter.
A: Can I now correct an impression that I left by answering affirmative to an earlier question?
Q: Certainly.
A: Now that I see this chapter, it is drawn in large part, of course, from the review that appeared in the American Review of Respiratory Diseases, but we added substantially to that; that is, authors Cain and Leaderer added substantially to it. So it was meant to be an update.
Q: Let's turn to Table 6.1 on Page 132. That table was titled "Established and Potential Health Effects of Involuntary Exposure to Tobacco Smoke," isn't it?
A: Yes.
Q: And in that table Dr. Samet has broken down the health effects into two groups, those being "Established" and "Potential"; is that correct?
A: I'm sorry, I looked at Figure 6.1.
Q: I may have said it wrong.

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A: All right.
Q: In that table under the heading "Potential Health Effects of Involuntary Exposure to Tobacco Smoke," Dr. Samet listed "increased respiratory symptoms in adults, reduced lung function in adults, increased risk for asthma, exacerbation of asthma, increased risk for cardiovascular disease, increased risk for nonrespiratory cancers, earlier age at menopause, increased risk for sudden infant death, and reduced birth weight"; is that correct?
A: That's correct.
Q: All of those were described by Dr. Samet as "potential," not as "established"?
A: That's correct.
Q: And you've already told us that you agreed with Dr. Samet's classification in this regard?
A: Yes, I do.
Q: Let's turn to Page 142 of that article. Under the heading "Respiratory Symptoms and Illnesses in Adults," the first paragraph, Dr. Samet wrote that "Consistent evidence of an effect of passive smoking on respiratory symptoms in adults has not been found"; is that correct?
A: That's correct.

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Q: And you agreed with that statement?
A: Yes.
Q: The first sentence, next paragraph, Dr. Samet wrote, "Neither epidemiologic nor experimental studies have established the role of ETS in exacerbating asthma in adults"; is that correct?
A: (Witness reviews document) I think in the same light, I agree with this sentence.
Q: Thank you. Let's turn to Page 148. I'm sorry, let's go back to 143 first. In the last paragraph of Dr. Samet's discussion of "Lung Function in Adults" he wrote, "A conclusion cannot yet be reached on the effects of ETS exposure on lung function in adults"; is that correct?
A: That's correct.
Q: And you agreed with that conclusion?
A: Yes.
Q: Now let's turn to Page 148. Page 148 is contained within Dr. Samet's discussion of lung cancer in this paper; is that correct?
A: Let me look back. I believe it is. Yes.
Q: In the second full paragraph on the page, beginning in the middle of the paragraph, Dr. Samet wrote, "However, most of the studies on involuntary

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smoking and lung cancer have small numbers of cases, and confidence intervals for the effect of involuntary smoking in the various studies would overlap. Variations in the results of the studies may also reflect random and nonrandom errors in the classification of exposure to ETS. In fact, assessment of exposure relevant to lung cancer appears more difficult than for other health effects of involuntary smoking."

Did you agree with Dr. Samet's observations in this regard? I should say, you did agree with them?

A: Yes, I did. It's an accurate reflection of the state of the literature at that time.

Q: One of the points that Dr. Samet is making there is the same point he made in the '87 paper, isn't it, and that is that the studies are still inconsistent with respect to whether they report an increase in risk and, if so, how large an increase?

A: Well, I think he is making a different point here. It's an observation on why they have variations in the risks that they report, and in part it is the difficulties of knowing with certainty what the surrogate exposures mean by way

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of actual dose. And so part of the variation is actually not surprising, given, one, the small numbers that he talked about in some of these studies, and, two, the imprecision in the actual extrapolation from the surrogate descriptors that stand for exposure to the actual dose.

Q: And so these are all in the nature of being explanations as to why the epidemiology may be producing variable results?

A: Yes. It's still totally consistent, though. I mean, it doesn't negate what you referred to in Table 6.1, that it had been established through a whole series of epidemiologic studies that there was a causal effect to respiratory cancer from passive cigarette smoke exposure --

Q: Well, Dr. --

MR. HOAG: Go ahead, you can finish your answer.

A: -- but there's variation in the magnitude of the exposure-response relationship.

Q: Dr. Spengler, I'd like for you to identify for me a single scientific report or review that says that the epidemiologic evidence on environmental tobacco smoke and lung cancer

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establish environmental tobacco smoke as a cause of lung cancer in 1991.

A: I think we said in this review that the Surgeon General's Report in '86 stated that.

Q: Have you reviewed the Surgeon General's Report?

A: Not for some time; not for ten years, no.

Q: You're not testifying that the Surgeon General based his conclusion on the epidemiologic studies of ETS and lung cancer, are you?

A: In part, yes. I mean, you have to look at the tenets of causality, and they include biological plausibility: Is there a response that's consistent with a dose-response effect with increasing exposure or dose? Is there a body of evidence that is consistent within itself? Were those studies designed properly? And I believe it was accurately reported when we said that the Surgeon General concluded that passive cigarette smoke was causal for respiratory cancers.

Q: I think the best procedure is we'll go back to the Surgeon General's Report in a little while and look at what he concluded and the basis for those conclusions. But let's look at the next

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sentence, the first sentence of the next paragraph in Dr. Samet's article that you've told us you agree with that states that "In spite of the variable epidemiologic evidence, ETS has been characterized as a cause of lung cancer."

Dr. Spengler, isn't what Dr. Samet meant there that the epidemiologic evidence is too variable to be the basis for the conclusion alone?

MR. HOAG: Objection to the form of the question. Objection to asking what the author meant when it's very clear what the author said by looking at the document itself.

Q: What did that sentence mean, Dr. Spengler?

A: Well, what it means is that you can have variability and also consistency. There's a consistency across the studies that were published at this time that when viewed in their entirety would support the conclusion that ETS is causal of lung cancer in nonsmoking populations. I think the best example, you wouldn't deny gravity is at work, but the effects of gravity over the surface of the Earth do vary, but yet the phenomenon exists. Just because one can measure it and measure variability or show variability in the measurement doesn't deny

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that there's a consistency in the relationship between masses.

So if you haven't been involved with epidemiology, this statement would obviously trouble you, but it doesn't – this comes at the end of a long review of a lot of the literature, and this isn't troublesome.

Q: Well, now the statement says that the epidemiology is variable; is that correct?

A: That's correct.

Q: And then it also says that in spite of this, ETS has been characterized as a lung carcinogen; is that correct?

A: That's correct.

Q: And then it goes on to discuss the IARC's and NRC's and Surgeon General's findings, correct?

A: Correct.

Q: And the IARC – at least Dr. Samet reports here that the IARC's conclusion was based on a number of lines of evidence, but the epidemiologic studies are not one of them, are they?

A: It doesn't say that it wasn't one of them; it says that they supported this conclusion on the basis of availability, the presence of a carcinogen,

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the availability of a carcinogen, the absorption of a carcinogen, and the nature of how dose-response relationships work for carcinogens. So I don't think the sentence says it excluded it; it said these other observations that actually I think are critical for anyone to believe the epidemiology. It in and of itself may not be sufficient unless the odds ratios were overwhelmingly large.

In this case the odds ratios aren't overwhelmingly large, they're rather modest by the way things go. So one looks to these other lines of scientific inquiry to support the interpretation of epidemiologic evidence.

Q: Dr. Spengler, have you ever read the IARC 1986 report that's referenced there?

A: No, not that I recall.

Q: And so you're not familiar with the conclusions that IARC reached with respect to the epidemiologic studies of spousal smoking and lung cancer?

A: No, I am familiar with it, because John Samet quoted it directly.

Q: Isn't it true, Doctor, that what IARC concluded in 1986 was that the epidemiologic studies

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of spousal smoking and lung cancer are consistent with either an increase or no increase of risk from ETS exposure?

A: You're telling me that's so, but I don't see it from this text that we're referring to here. It doesn't speak to that particular point.

Q: You simply don't recall whether that's what the report says or not?

A: Well, I said I didn't read the report, so I don't know whether it says that or not.

Q: I thought you also told me, "I am familiar with it"?

A: The IARC report?

Q: Yes.

A: No. I said I was familiar with the NRC report and the Surgeon General's Report.

Q: You must have misunderstood that question, but that's okay, we clarified it.

Let's turn to the next page, Page 149. In the first sentence of the last paragraph on the "Lung Cancer" section, Dr. Samet writes that "The extent of the lung cancer hazard associated with involuntary smoking in the United States remains uncertain, however." And you agree with that, don't

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you, Doctor?

A: I didn't quite keep up with you; I'm trying to look for it. Where was this?

Q: The first sentence.

A: Oh, here it is.

Q: Last paragraph.

A: "The extent of the lung cancer hazard." That's true.

Q: Let's turn to the next page, Page 150, which includes Dr. Samet's discussion of ETS exposure in nonsmokers and nonrespiratory cancers, and let's turn to the last paragraph of that section. That paragraph reads, "These associations of involuntary smoking with cancer at diverse nonrespiratory sites cannot be supported readily with arguments for biologic plausibility. Increased risk at some of the sites, for example, cancer of the nasal sinus and female breast cancer, have not been observed in active smokers (U.S. DHHS 1982). In fact, the IARC has concluded that effects would not be produced in passive smokers which would not be produced to a larger extent in active smokers"; is that correct, Doctor?

A: That's read correctly, yes.

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Q: And that's essentially the same conclusion that you reached in your 1987 review with Dr. Samet; is that correct?
A: It almost looks verbatim.
Q: And I take it, then, that in 1991, you still agree with Dr. Samet in this regard?
A: Yes.
Q: Doctor, do you still have the letter from Mr. Rosenblatt that has been serving as your expert disclosure statement in this case?
A: Yes.
Q: In the first sentence of the last paragraph of that letter where Mr. Rosenblatt wrote that "Dr. Spengler will further testify that it is the consensus of the worldwide scientific and medical communities that exposure to involuntary tobacco smoke in airline cabins causes disease," is that read correctly?
A: Yes.
Q: Are you prepared to testify on that topic?
A: Yes.
Q: What diseases have the worldwide scientific and medical communities concluded that ETS exposure in airline cabins causes? And I'll ask you further,

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as you identify those diseases, will you please tell me -- identify any scientific literature or other type of publication that you believe embodies or reflects that consensus.
A: I'll try. I think the table that you referred to in Chapter 6, which is Table 6.1, talks about the relationship between ETS and a variety of health outcomes that differentiates established and possible.
Q: Is that Table 6.1 on Page 132?
A: Yes. "Established" and "Potential." It reflects the consensus of the scientific and medical community as to the effects of involuntary exposures to tobacco smoke.
Q: And that would be current as of today; is that correct, sir?
A: No, it might be modified. I would suspect that some of the disease endpoints that are in "Potential" may actually be moved above the line and put into the "Established" categories. But I couldn't specifically identify and cite that literature. But, for example, "increased risk for cardiovascular disease," I believe the body of literature there has been expanded since 1991 and

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that I would be properly characterizing the majority of scientific and medical experts who are familiar with this literature saying that it has been established.
So to answer that part of your question, would this table exist today, I say with modifications.
Q: And you identified moving cardiovascular disease up.
A: Yes.
Q: Anything else?
A: I'd have to go back and look at both reduction in lung function and asthma; there has been some new work done on those topics. But let's put that aside, because that's not, I don't think, the essence of -- it might be to you, but it's not the essence of my remarks here. Where was I? I lost my train of thought.
All right. So that's the body of evidence, in summary, of the health evidence relating ETS exposures, involuntary exposures, and the likelihood or high probability of these outcomes.
Q: On this table, I believe your testimony, then, is that you would not move up "increased risk

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for nonrespiratory cancers" at this time?
A: No, what I would do in the intervening time before the trial would be to review the additional health literature on a few of these topics, such as asthma, such as respiratory symptoms in adults, and cardiovascular disease, and possibly nonrespiratory cancers. And sudden infant death there's been more work done. And I'd attempt to look at that evidence, possibly discuss it with John Samet, and see if we would reorder this table.
Q: Are you telling me that, as you sit here today, you would require further work to provide opinions on all those topics that you just identified?
A: Yes.
Q: Now, I want to make sure that you understood my question. Your disclosure statement says that you would testify about the worldwide scientific and medical communities consensus regarding ETS exposure in airline cabins. I believe that the answer you just gave me was not restricted to airline cabins; is that correct?
A: Well, I was getting to that point.
Q: Okay, please go ahead.

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A: But you're right about that.
And to date there's been – let me back up. There's been more literature – excuse me – more research done on ETS exposures in airline cabins following the publication of the National Academy report that provides a basis for understanding the exposures to passengers and crew under the conditions where smoking was permitted, and that the scientific community familiar with ETS effects would be compelled to interpret the exposures that were documented in airplanes to tobacco smoke and its products, along with a knowledge of flight attendant work history, to draw the conclusion that it was very likely that there could be a causal relationship between those exposures and disease, period.

Q: I appreciate that answer, Doctor. In a number of ways, with respect to airlines specifically, you stated your answer very conditionally, since you said that the scientific community would be compelled to conclude that it was very likely that there could be. In other words, Doctor, today would you agree that there is no consensus existing in the worldwide scientific or

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medical communities that exposure to ETS in airline cabins causes disease?

A: Nobody has asked them that question.

Q: Doctor, I'm not the one that has offered this as evidence, the Plaintiffs have.

A: Right.

Q: I want to know, you're telling me that – I think you're agreeing with me that such a consensus does not exist today.

MR. HOAG: Objection.

MR. FURR: Let me finish my question, please.

Q: But that there would be one if they only considered the issue; is that what you're telling us?

MR. HOAG: Object to the form of the question. Object to telling him what he just said. Object based on the fact that he's already asked and answered. The fact that you don't like the answer that he gave, that you may disagree with the answer you gave, may be a problem for you, but he answered the question.

MR. FURR: I don't think I'm going to disagree with the answer when he finally answers the

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exact question.

A: Well, you can argue what the first question is –

Q: Let's start over.

A: I thought I was answering that question. Now you start over by posing the question a different way or restating it.

Q: Would you agree, Doctor, that as we sit here today, there is no consensus in the worldwide scientific and medical communities regarding whether ETS exposure aboard airliner cabins causes disease?

A: I guess if you were strictly interpreting the words, I'd have to come back and say, a legitimately constituted, fully representative organization of science and medicine that have knowledge in this field have never made a proactive determination about the etiology of various diseases with respect to exposures in airplanes.

Q: Okay, thank you.

(Recess)

BY MR. FURR:

Q: Dr. Spengler, I want to go back briefly to the issue of whether there is a consensus of the worldwide scientific and medical communities about

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exposure to ETS aboard airline cabins. You told me that "a legitimately constituted, fully represented organization of science and medicine that have knowledge in this field have never made a proactive determination about the etiology of various diseases with respect to exposures in airplanes," correct?

A: That's what I said, yes.

Q: And so that's another way of saying, isn't it, Doctor, that as we sit here today, no such consensus exists?

A: No.

Q: It's not.

A: In my opinion, no. Do you want an explanation?

Q: Doctor, are you speculating as to what the consensus would be?

MR. HOAG: Wait.

Q: No, I don't want an explanation. I'm asking the next question. Are you speculating as to what the consensus would be if these worldwide scientific and medical communities attempted to reach one, or are you telling me that it's already been done?

A: No, I said I told – I'm not saying the

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latter, because I just said that hadn't been done. What I'm saying is, to repeat how I attempted to answer the question the first time, that given the fact that IARC, EPA, Surgeon General, National Academy of Sciences, represent a consensus within the scientific and medical community in their conclusions, that a subset of the diseases listed on Table 6.1 have been established as being causally associated with involuntary exposures to tobacco smoke. And so to the extent that the tobacco smoke exposures present on commercial airplanes when smoking was permitted are equivalent to or exceed those exposures that underlie the relationship to passive exposure to tobacco smoke in homes, then the consensus would be consistent.

Q: The consensus would be consistent?

A: Yes.

Q: If there were such a consensus, correct?

A: No, the consensus exists, as I would define a consensus. They exist as reflected in those publications that drew broadly upon the scientific and medical communities and extensive review of the health literature and exposure data. And that constituted a consensus to a causal relationship

between environmental tobacco smoke, involuntary exposure, and health consequences. There would be no reasonable scientist or physician that could possibly deny that exposures occurred on airplanes.

Q: That some exposure occurred on airplanes; is that what you're saying?

A: That's right.

Q: It's a yes/no: Did exposure occur on airplanes? Yes?

A: Correct. And that exposure was to environmental tobacco smoke.

Q: That's correct. But you're not telling us that the EPA, the NRC, IARC and the Surgeon General reached any specific conclusions about ETS in airline cabins, are you?

A: No, I'm not.

Q: And you're not telling us that the U.S. EPA reached any conclusions about diseases in adults and ETS exposure other than lung cancer, are you?

A: I don't know. I think the EPA document did include diseases in adults other than lung cancer in their association –

Q: Any nonrespiratory diseases?

A: I don't know; I'd have to go back and look

at it. That's why I said that the possibility exists that those diseases that are listed on Table 6.1 as potential could be reclassified.

Q: Can you point me to any scientific literature that has gone through the process that you've described and in fact reached the conclusion that ETS exposures aboard airliners when they existed was a cause of disease in nonsmoking crew?

A: Yes.

Q: What would that be?

A: The FAA report that measured a variety of environmental contaminants on some 90 flights. And in the analysis of that data, they conducted a risk assessment and concluded that there would be an excess risk of cancer, lung cancer.

Q: Is the FAA report that you're referring to also known as the Geomet report? Was Geomet the contractor?

A: It was done by Geomet. If I understood your question right, that would constitute – can you repeat your question to make sure I did and have responded properly.

Q: No, that responds. We'll talk later about the Geomet report and what the Geomet report did and

did not do and the basis for the conclusions of the Geomet report.

Can you identify any other studies, by the way, other than Geomet?

A: Not that I'm aware of.

Q: Geomet, of course, collected no new data on the risk from ETS exposure aboard aircraft, did it?

A: It did not.

Q: It did not. Is that correct?

A: Well, risk is made up of exposure and health outcomes, so I guess the answer is they did. I think what you implied by your question, did they collect any health data –

Q: That's correct, that's what I meant to ask.

A: They did not.

Q: They just took the epidemiologic studies of ETS exposure in other environments, assumed that they applied to the airliner cabin, and applied statistical models to those risks to reach certain estimates; is that correct?

A: Correct.

(Document marked Spengler Exhibit 4 for identification)

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Q: Doctor, you've been handed an exhibit that's been marked as Spengler Deposition Exhibit 4, which is a document entitled "Executive Summary, Aircraft Cabin Environmental Survey." Are you the John Spengler that's listed as the first author of this report?
A: (Witness reviews document) Yes.
Q: Is this an Executive Summary of the ABC 20/20 study that you mentioned earlier today?
A: Yes.
Q: Has there ever been a publication in the scientific literature on that study, other than this Executive Summary?
A: Parts of the study, yes.
Q: And are those listed in your CV?
A: I don't think so.
Q: Could you identify those publications for me?
A: I know that Harriet Burge and Mike Muilenberg, who both appear as coauthors of this Executive Summary, have presented the biological findings – excuse me – some of the biological findings at their scientific professional meetings. And I believe those meetings also included written

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extended abstracts. So in that sense, it would have been published in the scientific domain.
Q: Have any scientific articles on this study appeared in the peer-reviewed scientific literature?
A: No.
Q: Is it correct that ABC funded this study?
A: Yes.
Q: Would you tell us what was done in this study, please.
A: Yes. We put a sampling protocol together that included the parameters described in this Executive Summary. I can recite them again if you want.
Q: That's all right, no, you don't need to do that.
A: And we flew on some 22 flight segments, I guess, with the design to get near full load conditions and to attempt to answer the issue of public concern about the quality of the environmental conditions in aircraft cabins.
Q: This study was performed in 1993-'94; is that correct?
A: Yes.
Q: And so that would have been three to four

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years after the six-hour smoking ban was implemented in this country; is that correct?
A: That's correct.
Q: Did ABC tell you why they wanted this study conducted?
MR. HOAG: I'll just object as to questions about why a corporate entity called ABC wanted to do something.
MR. FURR: I asked him what he was told.
MR. HOAG: You just asked him what he was told.
MR. FURR: I asked him what he was told by ABC about why they wanted the study done.
A: John's interruption I think was useful, because it allows me to relate that we were approached by Dr. Timothy Johnson, who was a physician for ABC News, and asked if we would serve as consultants to a set of investigations into air quality on airplanes. And as we continued those discussions with him, it seemed reasonable that we should conduct the measurements ourselves. And they left it really up to us to determine which were the important parameters and conditions to investigate.
Q: And so the parameters and conditions that

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you investigated were in fact the ones that you thought were important to look at?
A: Yes.
Q: Is it correct that there was no smoking on any of the flights that you sampled – measured on?
A: That is correct.
Q: And you told us earlier that you have custody of the underlying data; is that correct?
A: Yes.
Q: My first question is one that I don't see addressed in the report, and that is, what did you tell us about the particulate levels that were measured in this study?
A: Nothing. We didn't measure particles in this study.
Q: You didn't measure particles in the study?
A: No.
Q: Why is that?
A: Because in the absence of cigarette smoking, we felt that the particulate levels would not be of primary concern or even secondary concern as an undifferentiated particle count mass. What we thought was more relevant is what is the biological component of airborne particles; namely, bacteria,

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fungi. And that's why we chose to focus on that
data as opposed to having the burden of carrying yet
another instrument. It was a trade-off; we couldn't
do absolutely everything we wanted to do.
Q: We'll get into this in more detail later,
but what is your bottom line with respect to the
average respirable particulate level aboard
commercial aircraft in the absence of smoking?
A: It's 20 micrograms per cubic meter plus or
minus 10 micrograms per cubic meter. You can always
find exceptions to that, but that is what I've seen
as a typical value on measurements that I've made
subsequent to this study.
Q: Let's turn to the Executive Summary and
look at the first paragraph, the fourth sentence in
the first paragraph that states, "Most of us,
unaware of the real and documented hazards of
increased exposure to ozone and radiation, express
concern about the also real possibility of airborne
infection"; is that correct?
A: Yes.
Q: Doctor, it is your opinion that ozone
exposures aboard commercial airliners are real,
documented hazards; is that correct?

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A: Yes, with the caveat not all flights, so
long as it's not taken as a universal statement.
It's not a hazard on every single flight, because
we've documented plenty of flights where there is no
ozone.
Q: But it is an occupational hazard that
members of crews face, isn't it?
A: Under very specific conditions, yes.
Q: What are those conditions?
A: Well, I can tell you what I believe at this
point are associated risk factors for high ozone on
airplanes.
Q: Okay. Please do.
A: High latitude flights, high altitude
flights, flights in the springtime in the Northern
Hemisphere, and flights for which their catalytic
converters are inoperative or poorly functioning.
Q: And I guess the common factor for all these
issues that you've listed is that they tend to raise
the concentration of.ozone in the commercial
airliner cabin; is that correct?
A: In their sum, yes.
Q: Now, on Page 7 of this Executive Summary
you state that "A substantial fraction of the

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flights, between 10 and 20 percent, had either the
three-hour or flight-integrated ozone levels
exceeding 100 ppb"; is that correct?
A: Yes.
Q: What were the highest ozone levels that you
measured?
A: Let me straighten out a misimpression.
This Executive Summary included the work done for
ABC News, 20/20, and that additional study conducted
by Sue Ludwig under our direction. Just so that the
ozone measurements are not incorrectly attributed to
the domestic flights, all right.
Q: Okay.
A: And so in effect, I've answered that
question that you've posed in our discussion about
other studies that we had done when I talked about
the ozone measurements made on transpacific
flights. And again, to the best of my knowledge,
the highest levels were in the 200 parts per billion
range.
Q: Did you measure ozone in the ABC study?
A: No, we did not.
Q: With respect to transpacific study, you say
that "Prolonged exposure to ozone at these levels

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has been shown to reduce lung function and cause eye
and airway irritation"; is that correct?
A: That's correct. Actually, that paragraph
starts out with "in our separate ozone survey." So
it makes a distinction there.
Q: Now, you seem to be distinguishing the
transpacific flights from the domestic flights with
respect to the ozone exposures.
A: Yes.
Q: Based on the factors that you gave me a
moment ago as risk factors for increasing the ozone
concentrations in the airliner cabin, this would not
have to do, obviously, with the time of year that
the flight is made, because domestic flights are
made the same time of year as transpacific flights,
correct?
A: Correct.
Q: And it would not have to do, would it, with
how well the catalytic converter was functioning on
the airplane, because that would be a commonality
between the two types of flights, wouldn't it?
A: Yes, it should be.
Q: And so this would primarily be a function
of the latitude and altitude that the plane flies

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in?
A: Yes.
Q: And in the transpacific flight what altitude were those planes flying?
A: I believe we may have that information, I think we do, but it was typically 40,000 feet plus or minus 2,000 feet.
Q: What altitudes do commercial aircraft fly at between the coasts in this country?
A: Transcontinental flights?
Q: Yes.
A: 30,000 – 35,000 plus or minus 5,000.
Q: So very comparable to the transpacific flights, correct?
A: No, I think the transpacific are higher, in a systematic way higher. I think they reach a higher cruise altitude.
Q: 5 percent higher, 10 percent higher?
A: Well, that could make a big difference.
Q: Now, you made the point about the transpacific flights, that it's prolonged exposure to ozone as these levels that can cause reduced lung function and eye and airway irritation. You would agree, wouldn't you, that the flight attendants on

those flights would be in the group that has prolonged exposure to those levels?
A: Yes.
Q: You also state in that same paragraph that "Tests with animals show that chronic ozone exposure results in tissue damage, loss of elastic recoil and increased collagen deposition at the bronchiolar level," correct?
A: Yes.
Q: Now, what are the health consequences of those physiologic changes?
A: Simply stated, ozone has been shown to change the underlying architecture of lung tissue that is equivalent to accelerated aging of that lung tissue. So the implications would be a decrease in total surface area for air exchange, decrease in lung volume. So all the processes that we go through in life anyway, this would accelerate that.
Q: Those are processes that underlie development of emphysema, aren't they?
A: In part, yes, but that happens primarily in the lower airways. This could happen throughout the conductive airways as well as the lower airways.
Q: Let's go back to the first paragraph of the

Executive Summary. You said that the other real undocumented hazard that some of us are unaware of is radiation; is that correct?
A: Yes.
Q: Is this the solar and cosmic ionizing radiation that we were discussing earlier today, Doctor?
A: Yes.
Q: Now, in your ABC study, you didn't actually sample for radiation, did you?
A: No, we did not.
Q: Again, was this one of those decisions you made in terms of the trade-off on how to spend your money and time?
A: Yes, that's exactly right. Making those measurements is rather – it's complicated. It requires a more sophisticated set of instrumentation than we had available to us.
Q: Doctor, it is your opinion that further study needs to be done with respect to the ionizing radiation exposures of commercial flight crews, isn't it?
A: I would say yes. In fact, it is being done; the National Institute of Occupational Health

and Safety is doing such a study.
Q: Now, that study is limited to reproductive effects, isn't it?
A: I believe that's the primary outcome they are going to look at.
Q: That study is not looking at cancers or neoplastic outcomes, is it?
A: That's not – no, they're not designed to do that.
Q: Dr. Spengler, who are the experts in this country on the radiation exposures of commercial airliner crews; are you familiar with the people that you consider to be experts on that topic?
A: No, I don't know them on a professional basis, no.
Q: Can you identify them by name?
A: I would say – I was going to say Dr. Dave Moeller, who is a health physicist who knows an awful lot about this topic, as well as exposures to radon and natural background radiation. But to the extent that he's actually done active research on this area, I don't know.
Q: Are you familiar with or aware of the professional reputation of a radiation physicist

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named Bramish?
A: No.
Q: Excuse me, Barish, the same question. The
name is Barish.
A: Where is he from; where is he located?
Q: New York.
A: At NYU, or the Department of Defense,
Department of Energy?
Q: I'm not certain.
A: No.
Q: Let's turn to Page 3 of the Executive
Summary. In the "Results" section you say that
"During boarding and deboarding CO2 was
substantially higher than during cruise, with levels
of 2000 ppm to 2500 ppm being typical," correct?
A: Correct.
Q: Now, what were the highest levels of CO2
that you found, do you recall?
A: I know we had some short-term exposures
above 3,000.
Q: And what are the health and comfort
implications of those types of exposures?
A: Well, that's a good question. There
certainly could be the – this is an indicator of

decreased ventilation per person, because the
individuals are the source of carbon dioxide, with
one exception, that might be the dry ice that is
sometimes used on the plane. So what this would
imply is that other contaminants that were in the
air were being diluted to a lesser extent under
conditions of this elevated carbon dioxide. So the
implications could be an overall sensory perception
among the occupants that the air is unpleasant,
uncomfortable, and perhaps even inducing irritation.
Q: This morning when we were talking about
your work for Boeing, you told us that one of the
factors that stimulated the study was the complaints
that flight attendants and their unions continued to
make to their employers, to yourself and others; do
you recall that?
A: Yes.
Q: And what's the nature of those complaints?
A: They vary. They have a set of symptoms
that include lightheadedness, headaches, feeling
nauseous, to fatigue, excessive fatigue, to
increased frequency of respiratory illnesses and
sinus effects. So it's a broad range of symptoms
that might involve multiple organ systems.

Q: Is it fair to say they just complained
about not feeling well?
A: Like I said, some are very specific as to
what it is that they don't feel well from, and
that's specific symptoms, and some could be
characterized as a general malaise and excessive
fatigue.
Q: Do they continue to complain of symptoms
that you would associate with exposure to irritants?
A: In some cases, yes.
Q: Do you have any expertise in the health
consequences of the disruption of an individual's
circadian rhythm?
A: No.
Q: On Page 5 of the Executive Summary you
state that "All dust samples had measurable
quantities of cat and mite antigen," correct?
A: Yes.
Q: Now, are cat and mite antigens irritants,
Doctor?
A: No, they're allergens.
Q: Well, now, on Page 6 in the discussion of
cat and mite allergens in the second paragraph,
first full paragraph on the page, you conclude by

saying, "There is a plausible biological connection
that can be made between endotoxin exposure and some
respiratory irritation reported from passengers and
crew," correct?
A: Yes.
Q: And so maybe I should ask, does exposure to
cat and mite allergens elicit the same symptoms as
being exposed to an irritant in part?
A: They could.
Q: What are the consequences for an asthmatic
of being exposed to cat and mite allergens?
A: If they were positively reactive to those
specific allergens, that given a high enough
concentration, you could induce wheezing, bronchial
constriction, or even a full asthmatic attack.
Q: On Page 5 of the report, with respect to
VOCs you state at the bottom of the page that "The
concentrations of total non-methane hydrocarbons is
quite high and exceeds the concentrations associated
with sick building complaints of eye and mucosal
irritation"; is that correct?
A: That's correctly stated, but I probably
would not draw the same conclusion as we finish up
the paper.

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Q: And why is that?
A: There's been a lot written recently on the inadequacy of using a combined VOC index in sick building investigations, that one doesn't know the real causative effect for eye irritation and mucosal irritation until you really determine the composition of the various constituents. For example, in this particular case, airplanes, the dominant compounds in the VOC categories were acetone, isopropyl alcohol, and methyl alcohol, which are not very irritating in and of themselves, because they're so water soluble. In fact, some of them – all of them are produced to some extent by our own bodies anyway, our metabolism. So I'm not as sanguine about the use of this non-differentiating VOC indicator.
Q: Are those compounds that you just identified irritating in combination?
A: I guess at some level you could find them irritating. I think so far we stated that none of the concentrations that were found of the VOC concentrations were found even in the closely approximating threshold limit values established for occupational limits.

Q: Now is this considering them in isolation?
A: That's considering them in isolation. But I don't want to contradict myself. I fully believe that the effects of mixtures will be experienced well below threshold limit values of individual compounds. And I think that's actually been established fairly recently in chamber studies.
Q: That's one of the things I was going to ask you. When assessing the toxicologic profile of a complex mixture, isn't it thought that the mixture itself must be studied as opposed to the individual constituents?
A: I think you have to do both.
Q: A moment ago did you say that none of the – you said that none of the individual non-methane hydrocarbons that you measured exceeded a standard. What was the standard that you referred to?
A: I was referring to the occupational standards, OSHA standards.
Q: Isn't it correct, Doctor, that in real world environments, none of the constituents contained in environmental tobacco smoke exceed those standards either?

A: I don't know that personally; I'd have to go back and look at the literature.
Q: But I take it that since that's the analysis you applied here and drew some meaning from it, you would think that that was something worth looking at?
A: What's the mixture?
Q: The threshold limit values for the constituents of environmental tobacco smoke.
A: It's one point of comparison; it's not the only point.
Q: But by looking at that, you determined that perhaps the conclusions about the non-methane hydrocarbons that you were making based upon your airline cabin measurements needed to be rethought; is that correct?
A: I wouldn't say rethought; I said I was rethinking this with respect to new evidence in the literature that suggests that total VOC as a combined measure of all the VOCs in a mixture was not holding up as a good predictor of building complaints related to eye, nose and throat irritation. That's the part I was rethinking.
Q: And you said you attached some significance

to the fact that none of the concentrations for these individual non-methane hydrocarbons exceeded their occupational threshold limit values, correct?
A: That's correct.
Q: And that same analysis should apply also to environmental tobacco smoke, shouldn't it?
A: If I were looking at all the constituents in this setting or some other setting where tobacco smoke is being used, one point of interpretation would be to compare it to threshold irritation values compound by compound.
Q: Thank you.
(Document marked Spengler Exhibit 5 for identification)
Q: Dr. Spengler, you've been handed a document marked as Spengler Deposition Exhibit 5. It is a copy of some testimony before the Subcommittee on Aviation of the Committee on Public Works and Transportation, House of Representatives, in 1994; is that correct?
A: Yes.
Q: Is this at least part of the Congressional testimony that you were attempting to recall this morning when we talked about Congressional

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testimony?
A: No, I remembered this one rather clearly.
Q: You just couldn't recall what committee or subcommittee?
A: That's true. I didn't know it with respect to this testimony, and I didn't know it with respect to the testimony that we may have given at the conclusion of our National Academy document.
Q: Is the copy that you've been handed highlighted?
A: No.
Q: You are the Dr. Spengler who was testifying here, aren't you?
A: Yes.
Q: Do you recall who asked you to testify?
A: Some staff member.
Q: Now, the testimony that you gave was related in large part to the ABC study that we just discussed, wasn't it?
A: Yes.
Q: In the second paragraph of your testimony you begin by saying, "We are finding out that many of the recommendations we struggled with and thought very thoroughly about are still before us. They

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are still not being implemented. I think we are even in a better position with regard to empirical data to come back to underscore many of those recommendations"; is that correct?
A: Yes.
Q: What recommendations were you referring to there, Doctor?
A: Talking about the recommendations within the scope of this Airliner Cabin Environment National Academy report.
Q: The 1986 committee report?
A: Correct.
Q: And which specific recommendations?
A: Well, I think we would have to go back and look at those recommendations, but I think at the time we were talking about ground operations; we were talking about the need to understand ozone better; we were actually, I think, even talking about the need to understand radiation exposures better. And of course we pointed out that there had not been a systematic, well-designed study on the health of flight crew or passengers.
And so what I was referring to here is that this data, along with data from the Geomet report or

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the FAA report, and other published reports, such as the Wilson's article on radiation levels measured for Quantas, say that we're in a better position to underscore some of the conclusions and recommendations that we made.
Q: And which conclusions and recommendations are you in a better position to underscore?
A: I'd have to go back and refer to these.
Q: Go ahead and highlight them for us, please.
(Discussion off the record)
A: I'll give you some examples.
Q: Okay.
A: We had a conclusion about ozone, and we said that if FAA – "Therefore the committee suggests that FAA carry out a carefully designed program to ensure that cabin ozone concentrations comply with Department of Transportation regulations."
Another one on carbon dioxide. "The FAA standard is much higher than standards for other confined environments. The committee recommends that FAA review its carbon monoxide standard."
We had something on ventilation. "The

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committee found that if the lowest rates of ventilation permitted by current equipment design were used under conditions of full or nearly full passenger load, the result of ventilation rates would be at a minimum determined to provide acceptable air quality when smoking is not permitted and other contaminant sources are not present."
I don't see where the recommendation is here, I'm sorry. Then it says, "Recommended," "a data collection program that measures the airflow and contaminants on airplanes should be implemented." And they did respond to that particular recommendation with the Geomet report.
There are others that call for a health study. Here. "FAA should consider rulemaking that restricts exposures to pregnant flight crew and cabin crew members. In addition, FAA should investigate total radiation exposures of flight crew and cabin crew members through the use of a statistical sample of full-time employees and should require airlines to provide precautionary information to their flight attendants about radiation exposures." It says, "The committee recommends that FAA establish a program to monitor

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selected health effects on airline crews."
So I could go through, but what I was referring to is not only our study but additional studies provide more evidence that they need to examine the consequences to this occupational group.
Q: And these statements were made in the context of being four years after the six-hour smoking ban, correct?
A: Yes.
Q: Has the FAA acted on any of your recommendations regarding radiation?
A: I don't know who the primary sponsor of the NIOSH study is, but to my knowledge – I mean, they are going to measure radiation. I don't know if any other studies have been done.
Q: In your Congressional testimony you then continue to say in the next paragraph that "even after our efforts on the National Academy committee, we continued to do our own surveys looking at particles, ozone, carbon dioxide, temperature, humidity, and radiation aboard commercial aircraft"; is that correct?
A: Well, let's see. Radiation is here, but we didn't do radiation. I don't know why I said this,

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but that's what it says.
Q: So you did not – you have not done any further research on actual radiation exposures?
A: No, I did not; I have not. So I probably misspoke there.
Q: But you have continued to look at particle levels aboard commercial aircraft even in the presence of the six-hour smoking ban, correct?
A: Yes.
Q: Now, in the next paragraph you mention Sue Ludwig's ozone study on transpacific flights.
A: Yes.
Q: And you testified that, quote, "This is very important data you should take a look at," correct?
A: Correct.
Q: What did you mean to convey by telling them it was very important that they should take a look at it?
A: Well, I was conveying that she was going to speak later in this hearing and that I would hope that they would pay particular attention to her testimony.
Q: And why is that; why did you attach

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particular significance to her testimony?
A: Because it was really showing data that was contrary to the conventional wisdom.
Q: So is it fair to say that under certain conditions she was showing that ozone exposures aboard commercial aircraft are higher than previously thought?
A: Yes.
Q: And it's important because the health consequences of ozone exposure aboard commercial aircraft therefore may be more severe than previously thought?
A: No, I don't think that was the case. It was more to the point that given the set of symptoms, complaints that flight attendants had in terms of their respiratory irritation and eye irritation and fatigue and possibly their increased infectivity, susceptibility to infection, that exposure to ozone was a plausible contributor.
Q: The last paragraph, Page 132 of your Congressional testimony, you state that "Despite the prevailing assumption among the flight crew and passengers that they are at risk from illness, reproductive problems, and from cancer, we still do

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not have a thorough study of prevalence," correct?
A: Correct.
Q: So your testimony is there is a prevailing assumption among flight crew members that they are at a risk of developing cancer from their occupational exposures?
A: Yes.
Q: And you were making the statement in 1994 in the context of a six-hour smoking ban, correct?
A: Yes.
Q: Were you in fact referring to the risk of cancer from ionizing radiation?
A: No, it was both, it was that plus – but I think a lot of the – I think a lot of the concern was, again, not from ionizing radiation; I don't think there was a high level of familiarity with that issue among flight attendants. I could be wrong, but at least those that I talked to, that wasn't expressed as being high on their list of concerns. It was – I think the issue of primary concern was the intercontinental flights that still allowed smoking, and some members of the flight attendants' associations were still exposed under those circumstances to cigarette smoke.

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But I think that the general statement is true, one would want to know the prevalence of cancer by tumor type, and then from tumor type, you do your age adjustment and gender adjustment and other things and determine whether you have any excess in this population.
MR. DAVIDSON: You said "intercontinental" flights with smoking; did you mean "international" flights?
THE WITNESS: I'm sorry.
MR. DAVIDSON: Thanks.
THE WITNESS: I did misspeak.
No, I said "intercontinental," yes; not "intra," "inter."
MR. DAVIDSON: You meant overseas flights?
THE WITNESS: I should have said that.
MR. DAVIDSON: As opposed to cross-country flights?
THE WITNESS: Right.
Q: You also said that "We still do not have a thorough study of prevalence," correct, the bottom of Page 132?
A: Correct.
Q: And is this the type of study that you've

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talked about on multiple occasions today with respect to not actually knowing whether commercial flight crews in fact experience an increased incidence of any of the diseases that we've been talking about?
A: You're saying, is that currently the situation?
Q: Yes.
A: To my knowledge, it is.
Q: On the next page of your Congressional testimony you say that "This needs to be done because we don't know the frequencies of illnesses. And case-control studies of cancer among the cabin crew and the pilots are needed desperately"; is that correct?
A: Yes.
Q: What were you referring to there?
A: I was referring to – well, I was referring to the fact that here is a large population, in particular the pilots, for which there was lots of medical evidence in terms of their every six months physical fitness, flight fitness examinations, but yet once they retired, there was no follow-up of this population. And that it was possible to do,

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either from early diagnosis of tumors or from actual cancer death records or death records identifying cancers within this population, do a case-control study.
Q: Let me try to restate the point you're making, and you tell me if I've got it right. Is the point you're making the simple fact that we don't actually know whether flight crews experience an increase in serious chronic diseases like cancer compared to other populations?
A: That's correct; it's anecdotal.
Q: In the next paragraph on Page 133 you say, "I just want to point out that radiation has taken on a new significance. There is a recent article published by Wilson, et al., in Health Physics this month about a study which looked at radiation exposures to the pilots and crew of Australian Airlines. They indicated that the exposures have increased over the previous ten years"; is that correct?
A: That's correct.
MR. FURR: Let's mark this, and I'll hand it to you, and you can tell me whether this is the Wilson study that you were referring to.

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(Document marked Spengler Exhibit 6 for identification)
Q: You've been handed what's been marked as Spengler Deposition Exhibit No. 6, I believe. Is that the Wilson study that you were referring to in your Congressional testimony?
A: (Witness reviews document) Yes, it is.
Q: In your testimony you said that "Radiation has taken on a new significance." What did you mean to convey by that phraseology?
A: I meant – what I was conveying is that it may be more important than previously thought because of – looks like trends and exposures, which I believe, if I recount this article correctly, it was a combination of change in aircraft design, plus flying higher, as opposed to a real change in the radiation flux to the Earth. And the other reason is that these flight crews are flying longer in terms of their occupational profession, more years, so they have the potential of having higher dose.
Q: And therefore a higher health risk; is that correct?
A: Yes.
Q: In the abstract to the Wilson article they

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state that "By use of this method, annual
doses up to 3.8 mSv" – which I believe is
milliSieverts – "were estimated for international
crew members"; is that correct?
A: Yes.
Q: What's the health significance of that
dose?
A: Well, it's just slightly below the limit
that has been established for occupational exposures
for radiation workers. So that's one point that's
significant. That's the primary point.
Q: And how was that limit for occupationally
exposed radiation workers established?
A: I'm not sure.
Q: But that limit is not a limit that's been
established to assure that there will be no cancer
risk from the exposure; is that correct?
A: I believe it's been established by this
International Committee on Radiation Protection.
And periodically they review the literature on
radiation, ionizing radiation effects and cancer,
and where they feel it's necessary, they revise
their dose limits.
Q: Let's stick with the Wilson article for one

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moment. If you would go to the "Discussion and
Conclusions" section on Page 500. In the context of
discussing radiation exposure to commercial flight
crews, Wilson states, "It is recognized that there
is no dose limit below which absolute safety can be
guaranteed." Do you see that statement?
A: Yes.
Q: You agree with that statement, don't you?
A: Yes.
Q: Let's go back to your Congressional
testimony. In the same paragraph you say – after
talking about the Wilson article, you say, "By
extrapolation, and because of the kinds of routes
that American crews fly, the radiation exposures of
U.S. crews could be higher than their Australian
counterparts. They didn't draw that conclusion from
their results, but I do"; is that correct?
A: Yes.
Q: And how high do you think that the
exposures of U.S. crews may be?
A: How high? I don't know.
Q: Something that needs further study?
A: Yes.
Q: In that same paragraph, your last sentence,

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you say, "Even at 5 MSR" – or at least the
transcript says that – "Even at 5 mSv"?
A: It must be mSv.
Q: "Even at 5 mSv per year, the risk of cancer
is one in 4,000, which is actually higher than what
we require for SuperFund cleanups," correct?
A: Correct.
Q: What would you say the risk of cancer is at
3.8 mSv per year?
A: It's proportional to the ratio of 3.8 to 5.
Q: So it's a linear relationship, in your
mind?
A: Yes.
Q: By the way, how did you derive this
dose-response relationship for 5 mSv having a risk
of cancer of 1 in 4,000?
A: I didn't derive it; I think that it was
stated somewhere. It could be in the Wilson
article, or it could have been in the other
publications, such as the National Academy Report on
Biological Effects of Ionizing Radiation. So I'm
sure I got it. I'm sure there's a reference on it,
let's put it that way.
Q: As you sit here, do you believe that that

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is an accurate description of at least one part of
the dose-response relationship for ionizing
radiation exposure and cancer risk?
A: I hope it is; I hope it's quoted right.
Yes.
Q: Now, you've told me that you're not sure
how high the radiation exposures are for commercial
crews in American – in U.S. domestic flights,
correct?
A: Correct. Let me go back to the previous
question.
Q: Sure.
A: Because it's in this Wilson article on Page
501, at the end of that first paragraph on the left
column.
Q: Okay. So you took that dose-response
information from Wilson?
A: Yes.
Q: Dr. Spengler, how does the occurrence of a
solar flare affect the ionizing radiation dose that
commercial aircraft crew members receive?
A: It affects it dramatically.
Q: Can you quantify that?
A: No. It varies by the altitude and the

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intensity of the solar flare.
Q: When you say "dramatically," can it double or triple the exposure?
A: Yes.
Q: Can it result in exposure of 200 times the normal dose received by flight crews?
A: In some certain locations, I believe. Over the poles it could be.
Q: How frequently do flares occur, Doctor?
A: Large enough ones to disrupt or have a noticeable change in the flux of incoming radiation, fortunately rather infrequently. It's decades. It's in the order of tens of years, I think.
Q: But there are in fact several solar flares each year, aren't there?
A: Yeah, there are some. Some don't escape the atmosphere of the sun.
Q: Is it correct that there is actually some type of procedure in place by which commercial aircraft pilots are supposed to be warned about the occurrence of a solar flare?
A: That's my understanding.
Q: Is it also correct that in fact commercial aircraft pilot crews in the United States never

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receive those warnings?
A: I wouldn't know.
Q: You were accompanied in your Congressional testimony by Dr. Burge, correct?
A: Yes.
Q: We've spoken about her before today. Have you reviewed the testimony that she gave at the hearing?
A: I was there, but I did not review this.
Q: Do you recall whether you agreed with the testimony that she gave?
A: We don't find ourselves in perfect agreement, but we agree on most things, yes.
Q: Let's look at her testimony on Page 134, the last paragraph of her testimony. She states that "There is not one single study that measures or in any way documents health effects along with air quality measurements. All the studies we are hearing about only report measurements of specific pollutants. We haven't heard about any study that correlates measure of specific pollutants with health effects. Until we have this kind of data, we are not going to know whether or not the air quality in airplanes is actually safe."

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That was her testimony, correct?
A: Yes.
Q: Did you agree with that statement?
A: No.
Q: What don't you agree with?
A: It would be good to have this information, and I think it's perfectly consistent with the answers I had given earlier on how I related it in my testimony here. But it's not an absolute requirement to have health data collected simultaneously with pollution data to make a reasonable judgment as to the safety of this internal environment or any internal environment. For instance, if there were a fire on board, we don't need to stop and make a measurement and correlate it with coughing of people to know that it isn't safe.
So I would be more inclined to say that measurements that have been made under physical conditions that are typical or represent maybe the upper extremes of the flight experience, even in the absence of direct health data, are valid and useful. But that doesn't mean we shouldn't strive to get at the answer to the underlying question,

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which is, at the current conditions experienced by passengers and flight crew, is their health and comfort being adversely affected by environmental conditions that could be modified?
Q: Let's go back to the committee's 1986 report for just a moment. Now, the committee did recommend a ban on smoking in commercial aircraft for the reasons that we discussed earlier, correct?
A: Yes.
Q: And that recommendation was made to the Department of Transportation, correct?
A: Yes.
Q: And it's also correct, isn't it, the Department of Transportation responded to the committee's recommendations in a formal document?
A: I guess so; I don't recall.
Q: You are aware that the Department of Transportation rejected the committee's recommendation to institute a smoking ban, aren't you?
A: It wouldn't surprise me.
Q: Well, you know a smoking ban didn't occur, did it, in 1984?
A: No; it didn't occur immediately following

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the submission of our report, no.
MR. FURR: Let's mark this.
(Document marked Spengler Exhibit 7
for identification)
THE WITNESS: This says 1986.
MR. FURR: 1986, I apologize.
Q: Doctor, you've been handed an exhibit
that's been marked as Spengler Deposition Exhibit
7. That is a Report to Congress entitled "U.S.
Department of Transportation" dated February 1987.
Have you ever seen that document before, Doctor?
A: (Witness reviews document) No.
MR. HOAG: Do you have a copy of that?
MR. FURR: No. If he's never seen it
before, we won't ask any questions about it.
MR. HOAG: Can I keep it?
MR. FURR: The court reporter gets to keep
that one, but I'm sure she'll permit you to make a
copy of it.
Q: Dr. Spengler, you are aware that in
response to the committee's recommendations, one
thing that the DOT did was commission the study that
you mentioned earlier by Geomet; is that correct?
A: Yes.

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Q: In fact, in the acknowledgment of Geomet's
study, you are listed as having contributed to the
report; did you know that?
A: You reminded me that I did assist them,
yes.
Q: How did you assist them?
A: I think there were some long telephone
conversations discussing design of the study. I may
have reviewed draft documents as they were prepared,
I don't recall, and commented on those. But they're
revision and submission.
Q: When you reviewed draft documents, do you
recall expressing any strong disagreement with
Geomet's findings?
A: I don't recall. I guess – I think,
though, I think what happened was that I reviewed it
in pieces. And I don't recall ever – although I
could be wrong, I don't recall reviewing their risk
assessment components that were done after the fact,
obviously.
Q: When you say you don't recall reviewing
them, do you mean you don't recall reviewing the
risk assessment components prior to releasing the
study?

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A: That's correct.
Q: But you have reviewed those components
after the fact; is that correct?
A: Yes.
Q: You're aware, then, aren't you, that Geomet
found that the risks from exposure to cosmic
radiation were estimated to range from 45 to 1,026
premature deaths per 100,000 for cabin crew and
passengers who fly frequently.
A: Say what number range again.
Q: 45 to 1,026 per 100,000.
A: 1.26?
Q: 1,026.
A: Per 100,000 or 105.
Q: Right.
MR. HOAG: Do you have a copy of this
document that you're reading the numbers from so he
can look at it too?
MR. FURR: Sure, I'll be happy to. We're
not going to be marking this today, but I'll be
happy to let him look at it.
A: (Witness reviews document)
Q: Yes, all right.
A: They split out passengers and crew

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separately on ETS.
Q: Doctor, do you have an opinion as to
whether Geomet's risk assessment for radiation
exposure to the cabin crew is a reasonable estimate?
A: I can't comment on that at this point.
Q: And is that because you haven't thoroughly
reviewed the Geomet report?
A: Well, it's been some time, and I'd have to
go back and look at the levels that they have
measured and how they did the exposure estimates and
what did they use for a potency factor.
Q: Do you have an opinion on the overall
quality of the study done by Geomet?
A: They're competent researchers.
Q: Geomet found that the risk of developing
cancer from radiation exposure for crew members was
many times the risk of developing cancer from ETS
exposure for crew members; is that correct?
MR. HOAG: I object to the form of the
question. He doesn't have a chance to look at that
while you're looking at it, and just looking at a
portion of it before...
Q: Go ahead, Doctor.
A: You characterized it as being many times

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different?
Q: Why don't you quantify the difference that they found for us.
A: Well, the differences would be roughly a factor of three to 50. I guess they would be even larger – this is a little confusing, I have to say, because for the ETS they separately present the estimated premature lung cancer deaths for nonsmoking flight crew members, cabin crew members, and passengers, and then they lump both of those together for the radiation. So see what I mean, it's difficult to separate on a comparative basis.
Q: And so if they had restricted the radiation risk assessment only to crew members, the differences they would have calculated for risk of developing cancer from radiation compared to ETS would have been even greater, wouldn't it?
A: No. I think – let's just take the upper bound estimate. I assume that the upper bound for the radiation applies to an upper bound estimate driven by the cabin crew exposure, because they would get more exposure than frequent flyers, passengers. So their upper bound estimate is, say, 1,000. And the upper bound estimate for flight

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crew, nonsmoking flight crew for ETS would be, say, 20 – 10 – let's say 20. So that's a factor of 50.
Q: And so if Geomet's risk assessments are accurate, Doctor, wouldn't that mean that in any given case of cancer in a nonsmoking flight attendant, it is 50 times more likely that that cancer was caused by radiation exposure than ETS exposure?
A: No, no. I'm not an expert in this area, I'm going to say, but there's a big difference between the tissue density and cosmic radiations zipping through our body. So it has a more likely chance of damaging tissue or genetic structure through bone and other more massive tissue and less for lung tissue, where the ETS would be more targeted towards – if these effects, results hold up, they would be targeted toward lung cancer.
So, for example, that's why we called for these kinds of studies. You've got to be able to classify the tumor site. And pilots think that they have excess brain cancer. It's got to be documented. And that would make sense, that would be associated with increased radiation exposure. But if there turns out to be in a nonsmoking flight

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crew increased lung cancer, then it's more likely than not that that would be ETS.
MR. HOAG: Does that include skin cancer too, radiation?
THE WITNESS: It probably would, but it's not – that tissue is so thin...
Q: Doctor, are you speculating now?
A: I'm answering his question, yes.
Q: Don't respond to his question, respond to my question.
A: I already caveated my statement when you asked me the question, and I'd say, "Well, let's sort of think through the issues here," and that's how I would think about it.
Q: Doctor, are you aware that the International Council on Radiation Protection has determined that lung tissue is more susceptible to the induction of carcinogenesis from radiation than bone is?
A: Why would that be? Well, that might be the case, and I was only using bone versus lung tissue as a comparative statement for density of tissue. Brain mass has a higher density than lung tissue. And so for the same relevant whole body exposure to

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particularly alpha particles, you would get subsequently more damage in more dense tissue.
Q: Doctor, I take it, then, you're not aware, are you, that the International Council on Radiation Protection has determined that lung, breast and thyroid are the most sensitive human tissues to developing cancer from radiation exposure?
MR. HOAG: Object to the form of the question. It sounds like a double negative; I'm not sure.
MR. FURR: Well, I think the record will be clear that it wasn't.
A: Well, if that's the case, I wasn't aware of it, but it could be radioisotopes. There could be lots of reasons that are different here. There are different ways in which radiation causes damage.
Q: This is simply not a field that you're expert in; is that correct?
A: I said that at the beginning.
Q: Let's go back to the question that got us started down this road. Without respect to the type of tumor in an individual, isn't it the case that if the Geomet estimates hold up that in any given case of cancer in a nonsmoking flight attendant it is 50

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times more likely that that cancer was induced by
radiation exposure that ETS exposure?
MR. HOAG: Objection to the form of the
question. That is most definitely not what he has
said already. He said just the opposite about lung
cancer.
MR. FURR: I have no idea what you're
talking about.
MR. HOAG: Well, I don't care whether you
know what I'm talking about. I want to put this on
the record.
MR. FURR: Why don't you make your
objection and stop speaking and let him answer.
MR. HOAG: No, I want to make my objection,
because you are changing the whole scenario here.
He has answered totally differently than what you
said, you know it, and all you're doing is making up
a question to try to confuse him.
Q: Can you answer the question, Doctor.
A: Hopefully I answered along the lines of
which I answered the earlier one. And I told you I
would be speculating, but I would argue it the
following way, and I could just repeat what I said.
Q: Doctor, I'm not asking you to speculate;

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I'm asking you the simple mathematical likelihood,
based on the Geomet estimates.
MR. HOAG: Objection.
MR. FURR: I'm not finished speaking.
MR. HOAG: Well, he wasn't finished either;
that's my objection.
Q: Were you finished, Doctor?
A: No. I have a different answer for your
question now.
Q: Okay. Why don't you go ahead and answer
the question now, then.
A: So your question, I'm going to paraphrase
it, and that is, that given any generic cancer that
a flight attendant might have, regardless of what
tissue site, that one would mathematically calculate
that the probability of that cancer being attributed
to cosmic radiation versus ETS would be 50 to one;
that's your question?
Q: That is my question, yes, sir.
A: And I would answer it the following way. I
would say – I'd say no for the following reasons:
that the risk assessment done on the basis of the
Geomet study was done with these contemporary
measurements; that the expression of a cancer at any

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given time presumably is a reflection of prior year
exposures, and this might be many years of
exposure. And we know that the exposures that
flight attendants experienced years ago in their
commercial service was throughout the plane and,
I'll speculate, probably was higher in its total
dose, because the population's smoking was higher
and there was no physical separation of smokers and
nonsmokers. And that you know from the Wilson
article that there are trends in radiation that
would indicate that radiation exposure has increased
in time, as the new generation aircraft have been
certified to fly at higher altitudes. So that prior
exposures to radiation would have been lower.
So in answer to your question, I would say
that it would not be 50 to one; it would be less
than that.
Q: How much less?
A: I don't know. First of all, I wouldn't
even do the calculation. But I just give that long
explanation of how I would think through the problem
and under the constraints of a hypothetical which
would be presented. But logically I don't think I
could accept that it would be 50 to one.

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Q: Okay. Doctor, do you still have your copy
of the '86 committee report?
A: Yes.
Q: Let's change our focus and begin talking
about ETS exposure.
MR. DAVIDSON: Is this a good time for a
short break?
MR. FURR: Sure.
(Recess)
BY MR. FURR:
Q: Doctor, as I said, I want to turn now to
talking about ETS exposure to crew aboard commercial
airliners. I ask you to turn first to the committee
report at Pages 145 through 146 beginning with the
bottom of 145.
A: Yes.
Q: Now, the committee attempted to compare ETS
exposures of flight attendants aboard airliners to
the ETS exposures experienced in homes of smokers;
is that correct?
A: Yes.
Q: Could you describe how the committee went
about doing that.
A: Yes, I can. As this describes in this

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paragraph and then continuing onto the next page, concentrations that look like typical values for respirable particle loads in the sections of planes where smoking was occurring was used as a reference value. So then the total dose that a flight attendant may actually inhale was calculated on the basis of 800 hours per year working and a breathing rate of 15 liters per minute, and that yielded an ETS integrated exposure of 1.8 x 105 micrograms per year.

And then by reference to concentrations that were reported in homes with smokers, a similar calculation was done for a hypothetical nonsmoking person living for about 70 percent of the total time in a house where one to one and a half packs of cigarettes were smoked per day, and a lower breathing rate to reflect resting conditions. And this yielded a number of 1.1 x 10[illegible] micrograms per year.

So on the basis of these comparative values, the committee recognized that the experiences of ETS exposure to flight crew members, primarily those working in the smoking section of the plane, would be equivalent to the nonsmoking

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spouse living in a house with a smoker.

Q: Okay. Let's break this down a little bit. First of all, the committee used RSP as a marker for ETS exposure; is that correct?

A: That's correct.

Q: We've been talking about them comparing exposures. In fact, they actually compared something slightly different than exposure, didn't they? And by that I mean, typically we think of, as I understand it – correct me if I'm wrong – that exposure typically means the product of concentration and time; is that correct?

A: That's correct.

Q: Here we're adding another factor for breathing rate; is that correct?

A: That's correct.

Q: And so is it correct that we are comparing quantities that could be described as potential inhaled exposures or something like that? How would you describe the actual comparison as conducted here, Doctor?

A: Well, if you don't like the term that is used, which is "integrated exposure," and one might say – you could say "inhaled dose," "annual

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dose" –

Q: Well, but it's really not a dose, is it, Doctor? Doesn't a dose connote you're talking about the concentration delivered to a target organ?

A: Well, there are attempts now in the literature to try to be more specific about these terms. In fact, that contribution in the literature occurred post the writing of this document. And I think if we were to have written this with careful consideration of those definitions, we might have distinguished between an exposure, which is a concentration of some substance at the boundary, at the interface to the body, that a dose is that material that passes that interface, and then the delivered dose or biologically available dose is something even further refined than that.

For example – and I do think it's relevant here – not all these particles stay in the lungs. Some go in and come out again. So that wasn't the point here, to be so refined about it. It was to do a first-level approximation to see if one could interpret the kinds of concentrations that were experienced in the smoking sections of airplanes and find its equivalence, if any existed, to exposures

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that were more familiar to us in other settings.

Q: I think the only thing that's important right now, now that we have your explanation, is that we find some language to use to describe what it is we're talking about, something other than "exposure," since it's exposure plus that we're talking about with the breathing rate. Could we call it "potential inhaled exposure"; would that be accurate?

A: I would call it "potential inhaled dose." You don't even need "potential," it's the inhaled dose. In other words, it passes the first boundary of the respiratory system, which is the nose.

Q: Okay, we'll call this "inhaled dose."

A: "Annual inhaled dose."

Q: So the committee attempted to determine the micrograms of RSP inhaled by a flight attendant over the course of the year and compare that to the RSP inhaled by a nonsmoker living at home with a smoker, correct?

A: Correct.

Q: And to do that the committee essentially multiplied three factors: the first being an ETS RSP concentration, the second a breathing rate, and

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the third a durational factor addressing number of
hours per year that the exposure occurred; is that
correct?
A: That's correct.
Q: Is this a reasonable way to attempt to
compare the residential exposures to exposures in
the airliner cabin?
A: It's one way of doing it.
Q: Well, do you have an opinion as to whether
there's a better way of doing it?
A: You said exposures in the residential
environment. I immediately thought of all possible
exposures to all sorts of things, and I could
imagine, although I can't express it right at this
second, that when you start talking about different
contaminants, you might think about it differently.
For instance, mite and cat antigen, you might think
of this comparison in a different way.
So if we just restrict our questions now to
ETS or RSP – I think this is a reasonable way, I
mean, for the purpose here. This was not a
definitive, probability-based estimate here.
Q: What was the purpose of this comparison?
A: The purpose was for the committee to

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demonstrate one component behind their thinking that
ETS exposures to flight crew may include the
possibility of discomfort effects, irritation and
health effects. And it was done, it's simply that;
it says, "Look, here you have some reasonable
estimates of duration of time flying, concentrations
experienced in the smoking section, and what might
be the breathing rates," and says, "Would this give
them the annual dosing rate for EST that would be
equivalent to living in a home with a smoker for a
year?"
Q: You also said that this was not a
definitive, probability-based estimate.
A: Yes.
Q: What did you mean by that?
A: Well, at the time we didn't have the
distributional data that was available after the
Geomet study. Let me back up. If this were to be
redone now, one might take central estimates for
each of these parameters and include a distribution,
in other words, a standard deviation, to them, and
do Monte Carlo simulations, which are commonly
available in commercial software packages now, to
look at the extent of overlap of these two

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distributions, the distribution being the estimated
annual dose, inhaled dose, to ETS.
Q: Okay. And you said that there was
information on those distributions available in the
Geomet report that was not available when this
comparison was made; is that correct?
A: On one distributional parameter.
Q: And what parameter was that?
A: That was on ETS level – excuse me –
respirable particulate levels in smoking sections of
planes and nonsmoking sections of planes.
Q: That would clearly provide a better
comparison than the comparison that was made at this
time, wouldn't it? By "this time" –
A: A different one. I don't know if it would
be better, but it would be different.
Q: Well, the Geomet approach would be better
for comparing the ETS exposure of the average flight
attendant to – I should say the RSP exposure of the
average flight attendant to the average RSP exposure
in a residence, wouldn't it?
A: Well, they wouldn't speak to the residence
issue.
Q: Fair enough.

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A: I would almost say yes to that, except that
one doesn't know that they're a representative
sample of flights, really a representative sample of
worker conditions – excuse me – work practice.
That's a little convoluted.
By that I mean, I would say that if I were
to do this over again, that taking that distribution
would be the starting point for the concentration
data, and I would go to work practice information to
get a distribution of flight attendants' actual
working conditions – excuse me – time.
Q: And by that are you referring to the same
type of information on the time/activity pattern of
flight attendants?
A: Yes.
Q: Now, the way Geomet got at that was to
assume that a flight attendant spent an equal amount
of time in the area adjacent to each row of seats in
the airliner cabin; is that correct?
A: I don't recall.
Q: If in fact that's the way that Geomet went
about that, would you consider that to be a
reasonable approach?
A: No.

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Q: Why not?
A: From my own observations, that's not the case; they don't.
Q: How does the flight attendants' time/activity pattern differ from that that I am representing that Geomet used?
A: My observation would be that they spend a higher proportion of their total flight time in the galley/service areas than they do around each seat.
Q: Okay.
A: I think I know the point you're driving at, and I know it's late, and I'm not going to defend this to the last decimal point; I mean, that's not the point. The point is that this was an order of magnitude estimate. Now, had it come out differently, had it come out in this simple calculation that flight attendants had 10 times less or 100 times less than someone with these reasonable assumptions in a home, then the committee would have thought about this differently.
Q: How would the committee have thought about it if it came out that flight attendants had three times less or four times less than the RSP exposures?

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A: I don't think it would have changed the final wording which was used, which we went over this morning, which was potential health effects; I don't think it would have changed that. It certainly would not change the phraseology to say it was causal, because there would be no logic to that.
Q: Doctor, I think you do know what I'm getting at here. What I'm getting at is the values used for these various parameters in the committee's comparison were not intended to represent the values for an average flight attendant, were they?
A: No, they were not.
Q: Because they assume that the flight attendant spends all of his or her working time in the smoking section; is that correct?
A: That's correct.
Q: And your own research, and I believe virtually all other research, shows that in airliners without recirculation that there are very different concentrations of RSP in the smoking section, the border section, and the distant nonsmoking section; is that correct?
A: That's correct.

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Q: And if one were to attempt to derive a comparison similar to the one found in the NRC report but designed to reflect the average flight attendant's average exposure in a year and compare that to a residential RSP exposure, you would need to find a way to weight the different concentrations of RSP in various sections of the aircraft with some information related to the time/activity pattern of flight attendants, wouldn't you?
A: Yes.
Q: That wasn't the intention of the comparison in the committee's report?
A: That's correct.
Q: Your intention was to make a rough cut at determining whether, I think you said, the exposures would be within an order of magnitude of each other; is that correct?
A: Yes.
Q: Doctor, if you'd look at Table 5.3 in the committee report. One of the studies cited in that table is your own study; is that correct?
A: Yes.
Q: When we talked about your original research on ETS exposure this morning, did we discuss this

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study?
A: These observations, yes.
Q: Is this the set of – I believe you described them as "anecdotal" observations you had made and not retained the data for?
A: That's correct. By "anecdotal," there was no attempt to have these represent a distribution that might be experienced on all 747s, for example.
Q: You just took measurements when you were on an airplane; is that correct?
A: That's correct.
Q: There was no intention that those measurements would represent anything other than the concentrations on the airplanes that you flew on?
A: Well, I did this enough times – well, no, I disagree with that as a conclusion. These were so predictable, and the experience was repeated so many times, not only on what is summarized here, but I think what actually is the experience of other people that have made these measurements, that they do stand for and do represent the range of values and the differences that are experienced in various sections of these aircraft, two types of aircraft, that permitted smoking.

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Q: But these were measurements of RSP and not RTS RSP; that's correct, isn't it?
A: This gets to be a terrible semantics problem in terms of the definition of instrument performance. But just leave it at that, yes, they are RSP values.
Q: Let's take a look at Table 5.3 and the information contained in that study from your personal communication. These data were not published in the peer-reviewed literature, were they?
A: No, they were not.
Q: Let's look at the first entry for 747s, for B747, for RSP. You indicate that the range was 10 to 50 mics per cubic meter in the nonsmoking section; is that correct?
A: Yes.
Q: And do you recall what the arithmetic mean would have been?
A: Over the duration of the flight, no, I wouldn't have that value.
Q: Now, earlier this morning didn't you tell us that the average RSP on nonsmoking flights is in fact a range very similar to this?

A: Yes, it is. Actually – yes, it is.
Q: Okay. So at least on nonsmoking flights, without recirculation, wouldn't you agree that there was very little difference in the RSP concentrations in the distant nonsmoking sections regardless of whether smoking was permitted?
A: I think you said something like the RSP on nonsmoking flights – is that what you said?
Q: No, I didn't mean to, if that's what I said. I did say it. I meant to just say flights without recirculation of air.
A: All right. The reason I clarified that, it is my observation that on flights without recirculation of air and nonsmoking, that the RSP levels are on the lower side of this range; for instance, 15 plus or minus 10 micrograms per cubic meter. But to be complete here, on the nonsmoking flights currently, where there is recirculation, the levels are slightly higher than that.
Q: Okay. Let's look at the top of Page 141 of the committee's report. The committee stated – right-hand column, top of the page – the committee stated that "In aircraft without air recirculation, passengers well into the nonsmoking sections, flight

crew members, and cabin crew members whose duties do not take them into the smoking sections are relatively unexposed"; is that correct?
A: That's correct.
Q: And they were talking about relatively unexposed to ETS; is that correct?
A: That's correct.
Q: Okay. Now let's turn to Page 45 of the committee's report, and the right-hand column, the next-to-the-last paragraph. The committee found that "In 1985, about 30 percent of the seat-hours flown by U.S. airlines were on aircraft with recirculation systems; is that correct?
A: I thought – oh, here it is. 145?
Q: No, Page 45. Page 45, right-hand column, the next-to-the-last paragraph.
A: "In 1985, about 30 percent of the seat-hours flown by U.S. airlines were on aircraft with recirculation systems."
Q: Now, another way of saying that is, in 1985, about 70 percent of the seat-hours flown by U.S. airlines were on aircraft without recirculation systems; is that correct?
A: Yes.

Q: Let's combine the last two issues. Would it then be correct to say that in 1985, in about 70 percent of the seat-hours flown by U.S. airlines, the crew members working in the nonsmoking sections whose duties did not take them into the smoking sections would have been relatively unexposed to ETS?
A: Well, no. Yes – leave it at that, with all those assumptions.
MR. HOAG: Let me just make an objection.
MR. FURR: It's a little late.
MR. HOAG: Well, it's more like an instruction to the witness. If your answer requires more to the answer, don't worry about how late it is. If you require more to answer the question fully, the lateness is not a problem. We want you to answer the question as completely as it needs to be answered.
A: Then I will amend my last answer.
Q: Go ahead.
A: In reality, there was more transient mixing of exposures in planes, because in two-class planes, and even three-class planes, first, business and economy, before the smoking ban there were multiple

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zones that permitted smoking. So it would be difficult for me to see – how to understand how a flight attendant would not get exposed to some ETS during the course of her onboard duties. Now, if they were assigned exclusively to first class and they worked in the front forward galley, then, yes, they would be relatively less exposed compared to flight attendants working in the aft galley in the smoking section.
Q: Doctor, the question wasn't whether on flights without recirculation the flight attendants had no exposure, but the thought that we put together with the committee's report was that they were relatively unexposed, a relative concept, correct?
A: I would say – you can't say "relatively unexposed."
Q: That's what the committee said.
A: Well, the committee should have said "relatively less exposed." Did they really say "unexposed"?
MR. DAVIDSON: 141, "relatively unexposed."
A: Terrible. Poor choice of words.
Q: Sort of like being relatively pregnant or

not?
A: Probably.
MR. HOAG: Did you have more to your answer to explain?
THE WITNESS: No.
MR. FURR: I think we understand him.
Q: We've talked briefly about the fact that on nonsmoking flights there is still a range of RSP values that can be measured; is that correct, Doctor?
A: Yes.
Q: And so in order to determine the ETS contribution to RSP in an airliner cabin, one has to find some way to subtract or sort out the RSP from other sources; is that correct?
A: That's correct.
Q: Now, some studies have attempted to do that by comparing RSP in airliner cabins for smoking flights to nonsmoking flights; isn't that correct?
A: Yes.
Q: In fact, that approach is similar to some of the work that you have done in which you have attempted to compare the RSP concentrations in homes with a smoker to the RSP concentrations in a home

without a smoker for the purposes of attributing a RSP contribution to ETS; is that correct?
A: That's correct.
Q: So you would think that's a reasonable approach to use to attempt to make the same type of comparison in an airliner, wouldn't you?
A: Yes, it's reasonable. I'm waiting for the "ergo."
Q: Well, I think we've got it already. You understand that that's the approach that Geomet used, don't you?
A: Like I said, it's been a long time since I read that document thoroughly. Are you talking about that's the approach they use in the risk assessment?
Q: In the exposure assessment.
A: I thought they showed the distribution of RSP without subtracting a RSP component on a nonsmoking flight or in a nonsmoking section; they just reported the data as it was, if I can recall properly. Now, the risk assessment, rightfully so, should have done a differential subtraction, I guess.
Q: Okay. Now, there are a variety of ways to

measure RSP, aren't there, Doctor?
A: Yes.
Q: One method is to measure the mass or weight of RSP collected on a filter; is that correct?
A: Divided by the volume of flow, yes.
Q: And that's a method that you have used in some of your studies, isn't it?
A: Yes.
Q: I take it it's a reasonable approach to take?
A: It's the best one we could think of at the time.
Q: Now, other methods include relating RSP concentrations to the scattering of light; is that correct?
A: That's correct.
Q: That's also a method that you've used in some of your research, isn't it?
A: Yes.
Q: Again, that's a reasonable approach to take, isn't it?
A: Yes, under some circumstances. I'd have to put caveats on that.
Q: Would it be a reasonable approach to take

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for measuring RSP in airliner cabins?
A: I would think so, yes.
Q: Let me ask you to describe – we've gotten at this indirectly a couple of times. As you sit here, what do you recall about the Geomet ETS exposure assessment?
A: I recall that I believe they used both methods, they use a gravimetric method, which was the filter method that you referred to, and they used an optical light scattering method.
Q: And do you recall that they used each of those methods in three sections of the airliner, those being the smoking, nonsmoking and boundary sections?
A: I don't recall that detail.
Q: You don't recall. You don't recall whether they conducted measurements on nonsmoking flights and subtracted those values from the values found for smoking flights in order to estimate the RSP contribution from ETS on smoking flights?
A: I know they did nonsmoking flights. I'm just getting this image of some of their data tables, and I don't recall that they actually did the subtraction – they may have – in the body of

the report.
Q: If in fact that's what they did, you would consider that a reasonable approach, wouldn't you?
A: Provided they paid proper attention and did the right sort of adjustment for contributing factors such as ventilation system or load factors. But in principle, it's reasonable.
Q: Doctor, in the committee's report they assumed that a flight attendant worked 800 hours per year in a smoking section; is that correct?
A: Well, exposed to these levels; I don't know if it says specifically in a smoking section.
Q: Let's go to 145.
A: Yes.
Q: It says, "We assumed a flight attendant worked 800 hours a year in the smoking section"?
A: Yes.
Q: And we talked about what the smoking section's component of that and what the comparison was or was not intended to accomplish?
A: Yes.
Q: Let's focus on the actual number of hours that a flight attendant works. Do you have any information with respect to what those hours are?

A: Yes.
Q: And what's your impression of how many hours a flight attendant works?
A: My impression is that they are limited to 80 hours or less per month. And many people don't work up to the maximum.
Q: And so this 800 hours per year might be a reasonable estimate of how many total hours per year a flight attendant works; is that correct?
A: Yes.
Q: That would be somewhere between 65 and 70 hours per month, wouldn't it?
A: That's what I think was the intention of this number.
Q: Assuming that a flight attendant works 800 hours a year, even one that works in a smoking section of that aircraft all that time, that flight attendant would not actually have been exposed to ETS for 800 hours per year, would he or she?
A: It depends on how they clock the time, but I believe take away some hours for flight preparation and takeoff before the smoking light came on, and on the other end of descent and landing, it would be probably somewhat less than

this that they would be exposed.
Q: But for those periods you just described, a flight attendant either would not have been exposed or the exposure would have been lesser, correct?
A: Well, it would have been more. No, I think it wouldn't be those two points, it would be primarily the point that – it could be both, let's put it that way. Their smoking may not be permitted and may not have occurred for that full 800 hours, plus they may have been out of that smoking section for short periods of time, plus the levels vary. Sometimes they're higher than this number; sometimes they're lower than this number.
Q: Okay. With respect to the breathing rates used in the committee's comparison, do you believe that those are reasonable breathing rates to use for flight attendants?
A: I think they're reasonable.
Q: Isn't that a breathing rate that correlates to light work?
A: I think that was the intention of choosing that value.
Q: In fact, flight attendants spend some percentage of their time sitting down, don't they?

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A: Yes.
Q: So for that period of time they spend sitting down, the breathing rate would actually be lower?
A: Yes, it should be.
Q: In the committee's comparison, the committee assumed that a nonsmoker would spend approximately 70 percent of their time in their home; is that correct?
A: Yes.
Q: In a number of your studies, you've suggested that that's a reasonable number, haven't you?
A: Yes. Although I'll say, in honesty, if I were to do this over again or have this done over again by anyone, it would probably be more realistic to use the currently available national time/activity data. But that wasn't available at the time.
Q: Do you know what that data shows?
A: It shows something in excess of 60, but if I round it off, I would pick 60 percent.
Q: Okay.
MR. FUHR: Would you mark this.

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(Document marked Spengler Exhibit 8 for identification)
Q: Dr. Spengler, you've just been handed an exhibit marked Spengler Deposition Exhibit No. 8; is that correct?
A: Yes.
Q: Do you recognize that exhibit?
A: (Witness reviews document) Yes.
Q: What is that? It's a very flattering picture, but in general, what is that?
A: It looks like someone got onto the web site for the Harvard School of Public Health and on that web site they have little clips of interest describing faculty.
Q: Did you create this document?
A: No. I might have done pieces or maybe my secretary clipped it from various things.
Q: In the first sentence of the second paragraph it says, "Our studies as well as others reveal that we spend 65 percent of our time in our residences"; is that correct?
A: Yes.
Q: Would you agree that that's the estimate that you would use if you were to do this again

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today?
A: Like I said, I would, depending on the purpose, one would probably be advised of the distribution from some citable national reference. So if I were to do point estimates, I would pick 65. If I were to do a full, more careful representation of the full distribution that might be experienced in homes and in airplanes, I'd approach it differently.
Q: Doctor, you have done – we went through them this morning – you have done a large number of studies in which you have, among other things, collected data that allowed you to compare the RSP levels in the homes of smokers and nonsmokers; is that correct?
A: Yes, among other things, yes.
Q: Those studies show that the difference in RSP between the homes of smokers and nonsmokers is between 20 and 40 micrograms per cubic meter, don't they?
A: As a generality, that's a fair range to characterize a broad difference between households.
Q: So the center point of that range being 30 micrograms per cubic meter, correct?

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A: Correct.
Q: So you would think that that was a reasonable point estimate to use to characterize the difference between the homes of nonsmokers and smokers with respect to RSP levels; is that correct?
A: Yes, yes.
Q: Dr. Spengler, if you were to today attempt to determine what were the average ETS RSP levels that flight attendants were exposed to when smoking was permitted on U.S. airliners, how would you go about it?
A: We're talking about retrospectively –
Q: Yes.
A: – what they were exposed to. I think one would have to try to make some differential between segregated smoking sections and non-segregated smoking sections.
Q: Let me interrupt you there. You're saying you would have to potentially develop different estimations for different periods of time?
A: Yes.
Q: Okay.
A: Again, it depends on what purpose you're trying to do this. If I answered this question and

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I ended up doing this, I might deviate from this method, you're asking me now, and this is what sounds reasonable to me now. But I probably would approach it in two ways. I would look at the available data, of which the Geomet is part, and a substantial part of that data, and use that as some of the distributional data for different sections of the plane, and I would attempt to refine the actual time/activity patterns of flight attendants. And if one wanted to calculate the annual inhaled dose estimates, then one would probably put some distributional data to the breathing, ventilation rates to reflect sedentary times and more active times.
Q: Okay. Other than the comparison reflected in the committee's report, have you made any effort to compare the RSP exposures of flight attendants on commercial airlines to residential RSP exposures?
A: No.
Q: Dr. Spengler, would you look at Page 21 of the committee's report, please. In the right-hand column, the last paragraph, the first sentence, the committee stated that "The scientific process involves the collection of reproducible facts, and

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scientific evidence is considered valid only if it is reproducible under similar circumstances or if lack of reproducibility can be explained"; do you see that?
A: Yes.
Q: Do you agree with that statement?
A: As a basic principle of scientific process, yes.
Q: Dr. Spengler, Appendix A to the committee report contains a model for estimating cabin crew RSP exposures; is that correct?
A: Yes.
Q: Now, you would agree that high quality measurement studies are preferable to modeling, wouldn't you?
A: Not in all circumstances, no.
Q: Why not?
A: Well, I think there's a financial situation that has to be considered, and also modeling may be the only way to predict some future condition, and so there's no alternative. But as a general statement, with all those factors being equal, it would be preferable to have empirical data than just modeling results.

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Q: Okay. Do you still have the exhibit that is Stanley Rosenblatt's letter that serves as your disclosure statement?
A: Yes.
Q: Turn to the second page. The last sentence says that your testimony will be based upon your review of tobacco documents, research and testimony; is that correct?
A: Yes.
Q: What tobacco documents will your testimony be based upon?
A: Other than the ones that were sent to me, I can't think of any. And quite frankly, even the ones that were sent to me, I don't see how they're relevant to my testimony.
Q: Okay.
A: I guess if you include it under the umbrella of "tobacco documents," the published papers that were of results that were conducted by scientific staff for the tobacco companies or funded by the tobacco companies, but that I would include under research.
Q: Well, that was going to be my next question. So is your answer the same with respect

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to tobacco industry research?
A: I don't know the full extent of that; I only know what has been reported in the literature.
Q: Well, as you sit here today, what tobacco industry research are you relying upon as a basis for your opinions?
A: I'd say no.
Q: As you sit here today, what tobacco industry testimony are you relying upon as a basis for your opinions?
A: I don't understand the question.
Q: Well, your disclosure statement says that your testimony will be based upon tobacco testimony, your review of tobacco documents, research and testimony. My question is, what tobacco testimony have you reviewed that you're relying upon?
A: I reviewed none.
Q: Okay.
MR. FURR: Let's take a break. I think I'm finished.
(Recess)

CROSS EXAMINATION
BY MR. DAVIDSON:

Q: Dr. Spengler, let me ask you first, you

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made an appearance before the ASHRAE Aviation
Subcommittee in January in Philadelphia, I believe
it was, and gave a presentation which was entitled,
as the minutes read, "Environmental Exposures During
Travel"; is that correct?
A: Yes.
Q: Is that the Boeing study that we've talked
about?
A: Yes, it is.
Q: And Mr. Furr asked you, in regard to your
disclosure of your testimony, he went through the
fact that you're going to rely, as you stated, on
your review of tobacco documents, research and
testimony. Do I take it this includes your own
research?
A: Yes.
Q: Does that include the Boeing study?
A: I don't think so, no.
Q: As you sit here today, you're telling us
then, sir, that you will be explicit, you're
explicit today, and you'll be explicit if you
testify in trial, that you're not going to rely in
any fashion at all on the Boeing work to support
your opinions?

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A: That's correct.
Q: Let's talk about your opinions for a bit.
You are going to testify regarding measurement of
tobacco smoke, and Mr. Furr has asked you
comprehensively about your own experience in regard
to measurement of tobacco smoke. You don't intend,
however, to testify or offer opinions as to dosage
flight attendants may have received of secondhand
tobacco smoke, correct?
A: Not unless it's asked of me, like it was in
this conversation.
Q: I'm sorry, which conversation?
A: This previous set of questions.
Q: Well, you haven't done it, have you? You
haven't done any work to determine dosage at this
point, have you?
A: Other than what we talked about on the
committee.
Q: Right.
A: Correct.
Q: But that's not really dosage, is it?
A: Correct, you're correct.
Q: Right. So you haven't done any dosage
work, and there's nothing in this disclosure that

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says you're going to do dosage work – that's all
I'm trying to establish – correct?
A: There's nothing in there – I don't want to
foreclose the option of doing that. I mean, we just
spent a long time here talking about how one might
approach this, and if that gives me an indication
that that's the line of inquiry that you'll pursue,
then I'd be silly not to try to do something to be
more responsive to that question.
Q: Doctor, let me just be very direct about
how the game is played.
A: All right.
Q: When we finish today, it's our position
that whatever you have testified here to today and
given opinions about, you're more than entitled to
do the same thing in front of a jury. We intend, as
we've done with Paul Halfpenny, who, I'm sure you
recall, established a record, that if you do
anything subsequent to develop additional opinions
beyond the scope of this and beyond the scope of
what you testified to today, fine, but we get to
come back and ask you about it in advance of trial.
So that we don't show up at trial and you say,
"Well, now since we saw you, Mr. Davidson and Mr.

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Furr, I've gone out and I've done a lot of dosage
research and here are my dosage conclusions." You
understand where I'm coming from?
A: Yes, I do, yes.
Q: That's the point of my question. And as
you sit here today, you've done no dosage work?
A: That's correct.
MR. HOAG: Obviously the issue of whether
or not you get a second chance at him if he does
dosage work is not something that he'll decide.
MR. DAVIDSON: Of course not. That's what
judges are for.
Q: And in the same vein as the discussion we
just had about dosage, the only exposure work you've
done is the work reflected in the airliner report –
I'm talking about ETS exposure work – correct?
A: No.
Q: What other exposure work as opposed to
measurement work?
A: I don't draw that distinction. How do you
define differently exposures versus measurements?
Q: Let me put it this way: Is there anywhere
other than the airliner report that your views on
measurement/exposure, as you characterize it, can be

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found, to ETS of course?
A: Well, let me just answer directly what I have done –
Q: Sure.
A: – so that you know. I would say over the last year since I received a new instrument, that's a lightweight, portable particle analyzer, I've taken to carry this on several flights. And for my own interest and curiosity, I would do transits on those flights by walking around all the sections and then staying in certain areas, and I've done this on flights to Europe and flights to Asia. So how can I not – that's part of my overall experience, and it's as relevant as any of the other measurements.
Q: Where is that data?
A: That data is in – it's downloaded off of this instrument; it sits in an electronic file.
Q: May we have that?
A: Let me get the traces, and I'd say yes. I don't think there's any problem with that.
Q: Any other measurements of exposure determinations; airliner, the personal work you've just described, anything else?
A: No.

Q: While we're in the "anything else" mode, let me go back to material that you have been provided by Mr. Rosenblatt or Mr. Hoag or anyone else from his office or any of the Plaintiffs in this case. We've agreed that you've received the letter which sends along the 42 documents that are bound, as I recall, correct?
A: Correct.
Q: You've received a book, correct?
A: Correct.
Q: I can't remember the title. You've received a copy of Dr. Burns' deposition, correct?
A: Correct.
Q: You've received no other copies of any other depositions, correct?
A: That's correct.
Q: Have you received a copy of the amended class action complaint?
A: I don't know if it's amended or not.
Q: Well, it's the actual complaint, the lawsuit.
A: Five pages or six pages?
Q: Well, no, actually, from memory, it's about 20 pages.

A: It is possible. There was a lot of repeat language in it, and I probably skipped through it, so it's possible. I do recall reading that.
Q: I just want to make very sure. Is there anything else?
A: Not that I can recall, no.
Q: And I'm not sure this question was asked. Have you assembled a working file for yourself to include documents other than the documents we've just discussed?
A: Not yet. I pulled off my shelf the FAA Geomet report and read it.
Q: And you've got your airliner report.
A: And I've got my airliner report. But I made no other attempt to pull together anything else.
Q: Do you intend to do so?
A: I think prior to my testimony I should reacquaint myself with a lot of our published work, yes.
Q: How may we learn what you decide is significant?
A: I could tell you that I would want to make sure that I was conversant, if I'm going to be

examined on the basis of my comparisons or opinion, that I properly relate the set of measurements and papers that we wrote.
MR. HOAG: Well, to the extent that it's work that he's published, you know what he's published; he's provided it to you.
MR. DAVIDSON: That's not the question. I think the Doctor understands exactly what my question is, which if he decides he needs to assemble additional material to assist him in testifying, he's making a subjective determination as to what's valuable or what isn't.
Q: And it may or may not include all your publications, correct?
A: That's correct.
Q: And it may certainly include publications and data in other studies that people have done; is that correct?
A: Yes, that's correct.
MR. HOAG: To the extent that it includes any of his publications, you've already had a chance to ask him any questions you wanted about any of his publications.
Q: What is your current relationship with

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Boeing, Doctor?
A: We have a contract with Boeing, which has
been extended for the completion of this report.
Q: Would I fairly characterize it as a
retainer sort of relationship?
A: No.
Q: So it's directed to the functions that you
have already performed and whatever additional
functions Boeing might see fit to ask you to do to
complete this study you're working on?
A: Well, let's see. We proposed some
additional work; they proposed - postponed decision
on that until our report is finalized. In addition
to that, they are starting up a new effort to
determine environmental factors affecting comfort
and health on airplanes. And they've had one
meeting - or several meetings of their internal
committee, and they asked me to attend one, and
under those circumstances they paid me a consulting
fee, which I then contributed back to the
university. So I'm not under retainer, but I
imagine, if they ask me again, I will work with them
under those conditions.
Q: You mentioned that you have - one of the

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sources of the information regarding flight
attendant complaints as to the current problems in
cabin air quality in the cabin atmosphere was your
discussions with flight attendants, correct?
A: And representatives of the flight
attendants' union.
Q: Can you name any of the flight attendants
you've individually spoken with as you sit here?
A: Other than Sue Ludwig, no, I don't know the
names of the others.
Q: And she's the person, the lady who's taking
a graduate degree in your program; is that correct?
A: No, she was a student in the night school.
Q: Okay.
A: And she has since graduated, and she's not
enrolled in our program.
Q: Since most of those measurements were taken
prior to May of 1994, why is that report not done?
A: I had - there were a couple of reasons for
it, but the short answer is that we are data rich
and analysis poor, and it just - we lag behind.
Q: I'm sorry. You mentioned Sue, whatever her
last name was.
A: Ludwig.

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Q: And who else, whatever other individual
flight attendants can you recall speaking with?
A: There were some flight attendants that I
had met on other occasions that were also present at
this ASHRAE meeting in Philadelphia.
Q: Is that the only ASHRAE TC 9.3 subcommittee
meeting that you've gone to, as best you can recall?
A: Other ones were attended by my staff
assistant that I didn't attend. I believe that's
the case.
Q: Okay. What about flight attendant union
representatives; who do you recall speaking with?
A: Ray Fenski.
Q: Fenster?
A: Fenster, sorry.
Q: What did Fenster tell you in regard to the
position of the AFA concerning cabin air quality and
cabin atmosphere?
A: He related the design and extent and some
of the initial observations from a survey that
they're currently implementing. I guess it's not
necessarily a survey; it's a reporting of
incidences.
Q: Health-related incidences?

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A: As he described it, it is anything from the
communication system doesn't work, to there's odors
on the plane, to it's too hot, and health-related
symptoms.
Q: You indicated in responding to Mr. Furr's
questions that radiation is certainly, if nothing
else, a significant health problem in regard to the
cabin environment potentially?
A: Potentially.
Q: Depending on the flight, the altitude, the
solar activity, et cetera, correct?
A: Yes.
Q: And do you know, Doctor, that EPA has
classified cabin and cockpit crew members as
radiational workers?
A: No, I didn't know that.
Q: If I told you that, that would underline
your concern, wouldn't it?
A: Yes.
Q: By the way, do you know Claude Thibeault?
A: Sounds familiar.
Q: He's the medical director for Air Canada.
A: Okay. I know of the name, and he was
supposedly going to show up at that meeting.

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Q: His white paper was actually presented for the Aerospace Medical Society. You've probably seen it.

A: Yes.

Q: And I just wanted to direct you to the fact that there had been a couple of Canadian radiation studies which are significant in this area also.

Just so we're clear on this Boeing study, you did present your findings verbally to the ASHRAE subcommittee in January, correct, your findings up through that date?

A: In part. I summarized some of the comparisons of a few of the parameters, and, like I said, some of the biological data is still in culture and actually hadn't been analyzed.

Q: And so that really there wasn't anything you withheld on the basis of confidentiality, you just presented what you felt was proper from a professional point of view to share with your colleagues, correct?

A: Our contract with Boeing has no confidentiality clauses.

Q: Okay. A couple of questions on the 20/20 Executive Summary; do you have it handy there?

A: Yes.

Q: I just want to confirm a couple of things. Page 5, the second full paragraph under the heading "Discussion." Now, this was written or this program was done in 1994, and in that paragraph you acknowledge concern regarding transmission of infectious viruses on aircraft, correct?

A: Correct.

Q: And I'm sure you're aware, Doctor, I believe, since you wrote this paragraph, that it has been confirmed by the – I'm sorry, I don't remember the name of the public health agency; I'm sure you know what I'm talking about – that there have been confirmed incidences of the transmission of TB between aircraft passengers and aircraft crew, correct?

A: My understanding, it's between passenger and passenger and between crew and other crew and crew and other passengers.

Q: I think you're absolutely correct. And that all occurred subsequent to your writing this paragraph, correct, the public health study that reached that conclusion?

A: The latter one where there was an

infectious flight attendant flying for several weeks in an infectious condition, that actually occurred prior to the writing of this thing, and I had seen the presentation of that work. So to answer the question, one of them occurred prior and the other afterwards.

Q: And the others that were reported on, actually the Aerospace Medical Society meeting in May of 1996 would confirm your concerns as expressed here, that it's important to pursue this issue further, correct?

A: Yes. I presume so; I don't know what was presented there.

Q: The other findings we're talking about. And the last thing I have in this area is on Page 6. I'm not sure we touched on this. The last sentence of the paragraph which concludes at the top of the page, this is a reflection of or a discussion of review of VOC measurements, and your conclusion is, "However, the eye and respiratory irritation of ethyl alcohol in combination with acetone, isopropyl alcohol, and other chemical compounds should be tested."

Do you know if testing has been done in

that area?

A: I'm aware of no testing done to date.

Q: Okay. You still hold the opinion that testing should be done, correct?

A: Yes, I do.

Q: Lastly, Doctor, just a few things back on your summary of what you're supposed to testify to. And I'm sorry, Jeff, help me here, because I may be doing something that you did while I was out of the room.

Mr. Furr asked you about this first sentence, the consensus of the worldwide scientific and medical communities, and I think your answers covered this question, but if not, help me. And that is, you cannot, as you sit here today, identify any person who you would vow to be in the worldwide scientific and worldwide medical community that would separately testify that the consensus is that exposure to ETS in airliner cabins causes disease, can you?

MR. HOAG: Object to the form of the question.

A: I think I could, yes.

Q: Who?

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A: I would think Samet might say this, John Samet.
Q: Anybody else?
A: We already have Dr. Burns.
Q: Dr. Burns is hardly an expert on ETS in airliner cabins, is he, Dr. Spengler?
MR. HOAG: Objection. You're arguing with the witness. He's answered your question, you don't like the answer, so you're arguing with him.
Q: You can go ahead and answer my question about Dr. Burns.
A: Let me go back, what constitutes an expert on ETS? He's someone who can look at the literature like anyone else can and say, "These are reasonably done measurements."
Q: Don't you think someone who holds himself out as an expert witness should have education and training in the field in question?
A: Well, if it's conditionally exclusive on all those sets of experiences and training, there aren't many people in the world that would ever be qualified.
Q: Well, let me ask you, Dr. Spengler. You're not a medical doctor; that's correct?

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A: That's correct.
Q: And you have not been retained in this matter to express medical opinions, correct?
A: That's correct.
Q: And just while we're on this subject, you're not an expert in ventilation systems, correct? You're not a ventilation engineer?
A: I'm not an engineer, but I know ventilation systems; I teach it.
Q: So you do consider yourself as having expertise in regard to opinions as to, for example, the relative efficiencies of ventilation systems?
A: Within the right context. One could always conjure up clean rooms that I might not be familiar with. But general principles of ventilation systems and how they work and how to make measurements of ventilation system efficiencies, yes.
Q: All right. And how about the configuration of airplanes? You're identified as someone who will testify regarding the configuration of airplanes. I assume this knowledge is gained from exposure to work in the areas that we've discussed today and exposure to airline cabin seating configuration charts; is that a fair conclusion?

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A: And schematic diagrams of their air conditioning systems and lots of hours of shared experience with Halfpenny and people at Boeing about how the systems work and onboard observations and reviews of the systems as the planes are being built. I'm certainly not a design engineer –
Q: That's fine. You're knowledgeable regarding what's there in regard to where the seats are and how they're arranged, and you're knowledgeable regarding the ventilation system in terms of where it is and how it works. But you're not a design engineer, and you're not a ventilation engineer, correct?
A: Correct.
MR. DAVIDSON: Let me just take a short break.
(Recess)

CROSS EXAMINATION
BY MR. HOAG:

Q: I just wanted to ask you a couple of questions related to, among other things, the document that was shown to you by Mr. Furr. The part that he read to you I believe is this paragraph, and I'll read it over again: "Estimated

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lifetime cancer risk to ETS exposure to be 12 to 16 premature lung cancer deaths per 100,000 nonsmoking cabin crew members." I'll stop with that sentence.
Do you know how they came up with that number of 12 to 16 premature lung cancer deaths per 100,000 nonsmoking cabin crew members?
A: Not in a lot of detail, no.
Q: Do you know in any detail?
A: Yes, I know they took the particle concentrations and derived an ETS fraction and applied a dose factor – excuse me, I should say a potency factor associated with a dose of ETS, and then extrapolated from there what the estimated population risk was. The details of where they derive that potency factor I don't know. And I think that the essence of it is understanding what assumptions they made in terms of the concentrations, the time/activity patterns of the flight attendants, and what the potency factor is.
Q: Do you know why the estimated range was within four, exactly 12 to 16 premature lung cancer deaths per 100,000?
A: That's rather unusual in terms of how

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narrow the range is, and it doesn't really discuss whether these are upper or lower bound estimates or the one standard deviation from the mean; it doesn't convey enough information.
Q: The next sentence says that risks from exposure to cosmic radiation range from 45 to 1,026 premature cancer deaths per 100,000 for cabin crew and passengers who fly frequently. Do you know how they came up with that estimated range of premature cancer deaths from people who are exposed to cosmic radiation?
A: They probably used the same mathematical construct, and that was to get a range of estimates for their exposures and the time/activity of the flight attendants and applied a potency factor to it, and the range probably reflects in part uncertainty in terms of what the exposures are. Some people would get very little and others would get quite a lot. But, again, this doesn't convey whether these are upper or lower bound estimates.
Q: Are you able to ascertain by this document what type of cancer or what types of cancer were included in that 45 to 1,026 premature cancer deaths per 100,000 estimation?

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A: No.
Q: Do you know what types of cancer are likely to result from exposure to cosmic radiation?
MR. FURR: Well, I'll object. He's already testified that's outside the scope of his expertise.
A: I think – well, I don't know. I don't think any specific cancer type could be absolutely excluded as having a potential causality due to cosmic radiation. It could affect DNA in any tissue of the body.
Q: Do you know whether skin cancer is caused by cosmic radiation?
A: No, I don't know. It's caused by solar radiation. I don't think it's caused by cosmic radiation.
Q: Do you know whether brain cancer is caused by cosmic radiation?
A: It certainly would be.
Q: Why do you say that?
A: Because cosmic radiation includes a step of ionizing particles which effectively could damage DNA.
Q: By looking – not even just by looking at

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this – do you know how many of that estimated range from 45 to 1,026 included brain cancer?
A: No.
Q: Do you know how many of that estimated amount included any type of cancer?
A: No, I don't think they specified tumor type.
Q: Is one more likely or less likely to contract brain cancer from cosmic radiation or lung cancer from cosmic radiation?
MR. FURR: Objection.
A: Well, based on my rather simplistic analysis, I would say it's more likely to have brain cancer initiated by cosmic radiation than lung cancer.
Q: Why is that?
MR. FURR: Objection.
A: Because of the density differences of the mass of the tissue.
Q: I want to refer you to Page 136 of "The Airliner Cabin Environment" report that you've looked at a number of times today. It's not an exhibit which is incorporated as an exhibit into this deposition; it's an exhibit in a prior

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deposition we took of –
MR. DAVIDSON: Halfpenny, Paul Halfpenny.
Q: Table 5.2 referring specifically to NNK, which there is a reference on the left-hand corner to NNK. What is "NNK"?
MR. FURR: Objection.
A: NNK is footnoted as Footnote b to this table, and it refers to methyl nitrosamine and some additional compounds, pyridyl-butanone, which is a nitrosamine.
Q: Based on your review of the literature, do you have an opinion as to whether or not NNK is a carcinogenic compound?
MR. DAVIDSON: Objection, leading.
A: I believe it is an established carcinogen. I don't know the specific classification that IARC would give it, but it's considered in occupational settings as a carcinogen.
Q: Now, in the table in the middle it says "Total Emission in Mainstream Smoke," and next to "NNK," what does that number represent?
A: This represents the mass of emissions in micrograms per cigarette that is involved in the mainstream smoke, which is that portion of the

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cigarette that would be inhaled.

Q: And the next portion of that table reads "Sidestream-to-Mainstream Total Emission Ratio." Can you explain what that means.

A: Yes. These results I believe came from smoking tests done in a chamber where sidestream smoke was captured separately from mainstream smoke and the total mass in those two fractions per cigarette were ratioed.

Q: What was the ratio when you compared sidestream smoke to mainstream total emission?

A: For this compound?

Q: Yes.

A: The ratios ranged from 1.1 or 10 percent higher to 4.1 – excuse me. I'll restate this.

They range from equal values – in other words, some studies showed that they were equal in their emission ratios – to other studies showing that they were four times higher in the sidestream smoke.

Q: So some showed that the sidestream smoke was four times higher?

MR. FURR: Objection, leading.

A: That's correct.

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Q: Do you know why the sidestream smoke was as much as four times higher for NNK as compared to the mainstream smoke?

MR. FURR: Objection.

A: Yes. The studies that describe these experiments as well as the summary of these experiments in various documents, relate to the enrichment factor; that is, that the sidestream of higher emissions per cigarette than the mainstream smoke for some compounds is the result of the pyrolysis of the tobacco under two different conditions.

Q: When you say "pyrolysis of the tobacco," what do you mean?

A: Primarily different temperatures of the burning flame tip of the cigarette. The sidestream, the flame temperature is lower during the mainstream phase of smoking.

Q: Hypothetically, if you knew that there was research indicating that the NNK nitrosamine level from cigarettes – from each cigarette was as much as 12,000 nanograms per cigarette, would that have changed – in your opinion, would that have changed whether or not the conclusion of the report used the

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word "potential" rather than "established"?

MR. FURR: Objection.

MR. DAVIDSON: Objection, leading.

MR. FURR: Calls for speculation by this witness regarding consensus of other people.

Q: You can answer it.

A: I'm not sure I fully understand the question. You said if I were shown some data or I were told that what, that 12,000 nanograms of NNK were emitted from what, the cigarette?

Q: From one cigarette.

A: From one cigarette. Mainstream or sidestream?

Q: Sidestream smoke.

A: Sidestream smoke. According to this data, the highest number that had been reported previously according to these references would have been something on the order of 4,000 nanograms of NNK in sidestream smoke. So if I were told that someone really had measured 12,000 – and this ratio could be incorrect, because you don't know what the reference base is. I guess if you reference it to the same cigarette, maybe the sidestream smoke was higher than these tests. So the ratio might

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actually stay the same, but the net contribution to the air would be higher.

I think it doesn't change my opinion a whole lot, because I think what I see from this table and the table on the previous page, which is for the vapor phase compounds, that many of these compounds that are produced from cigarette combustion appear to be enriched in the sidestream phase, and there is variability in these studies. So it would essentially just expand the variability for that particular compound. It might go from 1 to 1 to 12 to 1. It just means that more of it is available in the sidestream smoke.

And the way I look at all of this overall, I think it helps to support the biological plausibility that sidestream smoke is more irritating on a per mass basis and more carcinogenic on a per mass basis than mainstream smoke.

MR. HOAG: I don't have any other questions.

MR. FURR: I have just a few.

REDIRECT EXAMINATION
BY MR. FURR:

Q: Dr. Spengler –

MR. DAVIDSON: Let me do this, Jeff, if I

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could, because I'm going to take a stab at getting the 7:30.

MR. FURR: So am I.

MR. DAVIDSON: Cool. I was just going to put something on the record about where we are, that's all.

Q: The sidestream smoke comparisons contained in Table 5.2 that Mr. Hoag was just referring to are for fresh whole sidestream smoke?

A: I don't know what the "hold" means.

Q: Whole, w-h-o-l-e.

A: Oh, whole. I still don't know what the term refers to.

Q: Okay.

A: But the point is, under these experimental conditions, as you appropriately characterize it, I understand that this is freshly generated, relatively undiluted sidestream fraction.

Q: The ratios that are presented in this table do not represent in any way the ratio of exposures for these constituents the people incur in the real world for environmental tobacco smoke, do they?

A: Well, they're not meant to represent the

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exposures people incur.

Q: Because environmental – strike that. Sidestream smoke is not environmental tobacco smoke, is it?

A: Well, it's a major component of it, yes.

Q: But sidestream smoke is diluted almost instantaneously as it is generated from the burning tip of the cigarette, isn't it?

A: It's diluted into the larger volume of air, yes.

Q: What is the concentration of NNK in environmental tobacco smoke under real world conditions?

A: What's real world conditions?

Q: An average condition in an airliner.

A: I could make an approximation of it based on these numbers by knowing the mass of particulate phase and doing a proportion of that mass based on these emission factors, but I don't know offhand.

MR. FURR: Okay. No other questions.

MR. DAVIDSON: For the record, we do not conclude, depending on what Dr. Spengler's future intentions are regarding preparation for his

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testimony. Obviously, as Mr. Hoag has pointed out, that's a matter for the judge to determine, not us.

(Whereupon, at 7:00 p.m. the deposition was suspended sine die)

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CERTIFICATE

I, John G. Spengler, Ph.D., do hereby certify that I have read the foregoing transcript of my testimony, and further certify that said transcript is a true and accurate record of said testimony.

Dated at __, this ____ day of _______, 1997.

Sworn and subscribed to before me this day of __________, 1997.

Notary Public
My commission expires:

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COMMONWEALTH OF MASSACHUSETTS)
SUFFOLK, SS.)

I, Anne H. Bohan, Registered Diplomate Reporter and Notary Public in and for the Commonwealth of Massachusetts, do hereby certify that there came before me on the 30th day of April, 1997, at 10:10 a.m., the person hereinbefore named, who was by me duly sworn to testify to the truth and nothing but the truth of his knowledge touching and concerning the matters in controversy in this cause; that he was thereupon examined upon his oath, and his examination reduced to typewriting under my direction; and that the deposition is a true record of the testimony given by the witness.

I further certify that I am neither attorney or counsel for, nor related to or employed by, any attorney or counsel employed by the parties hereto or financially interested in the action.

In witness whereof, I have hereunto set my hand and affixed my notarial seal this 5th day of May, 1997.

Notary Public
My Commission expires 1/17/2003

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March 14, 1997

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VIA FAX

TO: All Local Counsel of Record

Re: Norma R. Broin, etc. et al. v. Philip Morris Companies, Inc., et al.
Case No. 91-49738 CA (22)

Dear Counsel:

We are withdrawing Kenneth E. Warner, Ph.D. as an expert in Broin and substituting in his place John Spengler, Ph.D. who is available for deposition the week of 4/28 - 5/2 at Harvard Medical School in Boston.

Dr. Spengler is a Professor of Environmental Health and Director of the Environmental Science and Engineering Program in the Department of Environmental Health, at Harvard University School of Public Health. Dr. Spengler has conducted research in the areas of personal monitoring, air pollution health effects, aerosol characterization, indoor air pollution and air pollution meteorology. Dr. Spengler served as a member or consultant on various United States EPA Science Advisory Board Committees. Dr. Spengler has a Ph.D. in atmospheric science and a Masters in public health.

Dr. Spengler is expected to testify regarding the air quality in airline cabins and issues regarding environmental tobacco smoke in airline cabins. Dr. Spengler was the Vice Chairman of the Committee on Airliner Cabin Air Quality that published the study "The Airliner Cabin Environment -- Air Quality and Safety" in 1986. Dr. Spengler will describe all facets of the study and the unanimous recommendations of the Commitee that there be a ban on all smoking on domestic commercial flights.

Dr. Spengler will further testify that it is the consensus of the world wide scientific and medical communities that exposure to involuntary tobacco smoke in airline cabins causes disease. Dr. Spengler will further testify regarding the 1986 U.S. Surgeon General's Report on second hand smoke. He will further testify regarding the measurement of tobacco smoke in airline cabins, including the configuration of planes, the ventilation systems and all issues related to flight attendants' exposure to environmental tobacco smoke in airline cabins.

DEPOSITION EXHIBIT
Spengler 1
4-30-97 AB

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All Local Counsel
March 14, 1997
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Dr. Spengler's opinions will be based upon his education, training, skill and experience. It will also be based upon his personal research and his knowledge of the consensus in the world wide scientific and medical communities regarding second hand tobacco smoke or environmental tobacco smoke. It will also be based upon his review of tobacco documents, research and testimony.

Very truly yours,

Stanley M. Rosenblatt

SMR/rab

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Health Effects and Sources of Indoor Air Pollution. Part I[1-3]

DEPOSITION EXHIBIT
Spengler 2
4-30-97 AB

JONATHAN M. SAMET,[4] MARIAN C. MARBURY,[5] and JOHN D. SPENGLER

SUMMARY Since the early 1970s, the health effects of indoor air pollution have been investigated with increasing intensity. Consequently, a large body of literature is now available on diverse aspects of indoor air pollution: sources, concentrations, health effects, engineering, and policy. This review begins with a review of the principal pollutants found in indoor environments and their sources. Subsequently, exposure to indoor air pollutants and health effects are considered, with an emphasis on those indoor air quality problems of greatest concern at present: passive exposure to tobacco smoke, nitrogen dioxide from gas-fueled cooking stoves, formaldehyde exposure, radon daughter exposure, and the diverse health problems encountered by workers in newer sealed office buildings. The review concludes by briefly addressing assessment of indoor air quality, control technology, research needs, and clinical implications. AM REV RESPIR DIS 1987; 136:1486–1506

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Introduction

During this century, dramatic episodes of excess mortality caused by ambient air pollution convincingly established that atmospheric contamination by human activities can adversely affect health. In many countries, governmental regulations implemented in response to the adverse health effects of air pollution have resulted in strong trends towards improved air quality. As the hazards posed by ambient air pollution from conventional fossil fuels have diminished in some countries, the relevance of indoor air quality for health has become increasingly apparent. Studies of time-activity patterns demonstrate that residents of more developed countries spend on average little time outdoors (table 1) (1, 2). Thus, indoor pollutant concentrations are the predominant determinant of exposure for many pollutants and the only source of exposure for some. However, pollutants in outdoor air do penetrate indoors, and for some pollutants of current importance, such as ozone and acid aerosols, nearly all exposure, whether received indoors or outdoors, results from outdoor sources.

Research directed at indoor air pollution and its adverse health effects began in the late 1960s and early 1970s (3, 4). Investigation in this area was subsequently stimulated by concerns that reduced ventilation of buildings for the purpose of energy conservation would increase pollutant concentrations and lead to adverse effects on health. Consequently, a large body of literature is now available on diverse aspects of indoor air pollution: sources, concentrations, health effects, mitigation, and policy.

While many health effects of indoor air pollution remain controversial, epidemiologic and clinical research has identified some health effects that should be considered by chest physicians and other health care providers. The public has been intensely interested in the new information on indoor air pollution, particularly as it relates to such ubiquitous exposures as formaldehyde, environmental tobacco smoke, radon and radon daughters, nitrogen dioxide (NO_2) from gas-fueled cooking stoves, and smoke from woodburning fireplaces and stoves. Patients may turn to their health care

This is Part I of two parts; the second will appear in the next issue of the Review.

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TABLE 1

AVERAGE HOURS SPENT PER DAY IN VARIOUS LOCATIONS BY ADULTS IN 44 U.S. CITIES*

Location	Employed Men	Employed Women	Housewives
At home	13.4 (55.8)†	15.4 (64.2)	20.5 (85.4)
At work	6.7 (27.9)	5.2 (21.7)	— (0)
In transit	1.6 (6.7)	1.3 (5.4)	1.0 (4.2)
Outside	0.7 (2.9)	0.3 (1.3)	0.4 (1.7)
Inside other structures	1.6 (6.7)	1.8 (7.5)	2.1 (8.8)

* Based on data in tables 7-1.1 and 7-1.3, page 796 in reference 1. Time calculated for "outside" includes the categories "just outside one's home" and "in all other locations." Time calculated for "inside other structures" consists of the categories "in other people's homes," "in places of business," and "in restaurants and bars." The original data did not separate work into indoor and outdoor categories.
† Percentage of 24 h.

providers because of concerns about potential health effects of these and other indoor air pollutants.

The findings reported in this new literature also have wide-ranging policy implications (5). Evidence of adverse health effects of indoor air quality may require decisions and actions on consumer products, building materials and design, energy conservation practices, and regulation of smoking in public places. In the United States, the Environmental Protection Agency under the authority of the Clean Air Act regulates to protect and enhance outdoor but not indoor air quality. This agency has constructed a statutory framework for implementing ambient air quality standards and has devised a complex set of regulations for controlling mobile and stationary air pollution sources.

For some criteria pollutants, an encouraging trend of improving outdoor air quality has resulted. The number of locations exceeding the primary National Ambient Air Quality Standards for total suspended particles (TSP), sulfur dioxide (SO_2), carbon monoxide (CO), and lead (Pb) has decreased over the past decade. Even peak ozone (O_3) concentrations have declined in many locations. Of the 6 criteria pollutants, only NO_2 pollution has worsened (6). However, improvements in ambient air quality do not necessarily imply that human exposures to harmful pollutants have also declined. Indoor air quality is not directly regulated, and use of some sources of indoor air pollution, such as wood stoves and kerosene space heaters, is increasingly widespread. Low air exchange rates in newer homes and office buildings may also increase personal exposures. Thus, air quality policy designed to fully protect public health must address exposures to pollutants indoors as well as outdoors.

This review summarizes information on the health effects of indoor air pollution, with an emphasis on the data that are most relevant for health care providers and those concerned with public health aspects of indoor air quality. We have also focused on the indoor air quality problems of greatest public health concern at present and emphasize those for which new evidence has become available: passive exposure to tobacco smoke, NO_2 exposure from gas-fueled cooking stoves, formaldehyde exposure, radon daughter exposure, and the diverse health problems encountered by workers in newer sealed office buildings. The citations are based primarily on a literature search that extended through June 1986; selected references subsequent to that date have been cited.

Review articles (7–10) and several monographs (11–14) on indoor air pollution have been published, as well as a report by the National Research Council (15). Proceedings of meetings on this topic have also been published (16–24). Numerous sources on the health effects of ambient air pollution are also available, including a statement of the American Thoracic Society (25), reports on individual pollutants by the National Research Council, and the criteria documents prepared periodically by the Environmental Protection Agency.

In this review, we initially consider the sources of indoor air pollution and information on personal exposures to indoor air pollution. Subsequently, for each of the major pollutants, we review the concentrations in indoor environments and the health effects. We conclude by briefly addressing indoor air quality assessment, control technology, research needs, and clinical implications.

Indoor Air Pollution: Sources and Exposure

Introduction

In this section, we highlight information on the sources of those pollutants that have been, or are, potentially associated with disease. We also review studies of personal exposures to pollutants. Concentrations of pollutants in indoor environments are described in subsequent sections on individual pollutants. We do not attempt to cover exhaustively the data on sources and exposures; comprehensive treatments are available in the report of the National Research Council (15) on indoor air pollution, in a review by Yocom (26), and in the proceedings of the Seventh Oak Ridge National Laboratory Life Sciences Symposium (24).

The health risks posed by air pollution are determined by the personal exposure of individuals to contaminants and not simply by pollutant concentrations in indoor and outdoor air. Personal exposures to air pollutants represent the average of the pollutant concentrations encountered in various environments with weighting proportional to the time spent in each location (figure 1). In more developed countries, studies of activity patterns have established the importance of the indoor environment in determining personal exposures (table 1) (1, 2).

The determinants of indoor concentrations vary among the pollutants. Levels may be influenced by outdoor levels, indoor sources, the rate of exchange between indoor and outdoor air, and other characteristics of the structure and its furnishings that influence pollutant dispersion and removal (15). Pollutants from

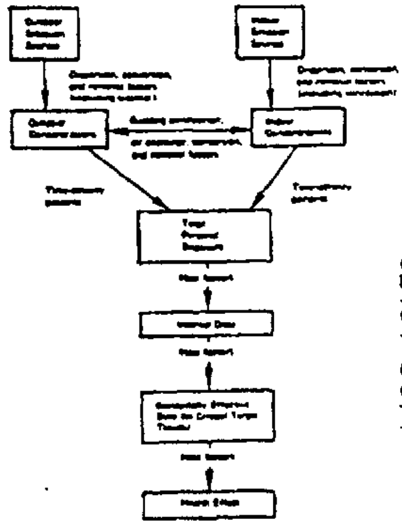

Fig. 1. Framework for considering the relationships among pollutant concentrations, personal exposures, doses of pollutants to target tissues, and health effects. (Reprinted with permission from reference 27.)

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outdoor sources can enter a building through mechanical ventilation systems and through the natural infiltration of air. The indoor concentrations of "outdoor pollutants" depend not only upon the outdoor concentrations but upon the rate of air infiltration, the reactivity of the contaminant, the efficiency of any mechanical filtration systems, and, for solids, upon the particle size and shape. For example, O_3, primarily an ambient pollutant, is a highly reactive molecule; it follows first-order decay kinetics when penetrating indoors and usually reacts quickly with surfaces. Indoor concentrations of O_3 are usually less than 50% of outdoor concentrations (28). Nevertheless, even small amounts of O_3 will be important in some indoor environments, such as art museums and locations with NO_2 sources. The concentrations of pollens, which are large particles from outside sources, decline substantially with increasing distance from doors and windows (29). Outdoor sources may lead to unusually high pollutant levels indoors if fresh air intake vents are improperly located. For example, increased CO levels have been measured in buildings with intake vents fed by air contaminated with vehicle exhaust from adjacent roadways or parking garages.

Increasing concentrations of indoor air pollutants have been of particular concern as ventilation rates have been reduced in newer structures. The 83 million housing units in the United States are diverse in character and some types are more likely to be associated with excessive indoor air pollution than are others. In new "tight" homes, air exchange rates during winter may average less than 0.5 per hour, while most conventional homes have average exchange rates between indoor and outdoor air around 1.0 per hour. Energy efficient "super tight" homes can be built with winter air exchange rates as low as 0.1 to 0.3 per hour. In comparison with conventional homes, mobile homes and prefabricated housing units have lower mean air exchange rates and are of smaller volume. These characteristics make them particularly liable to indoor air pollution problems. In many new office buildings, construction techniques and ventilation practices also lead to low air exchange rates and the potential for air quality problems.

Although most time indoors is spent at home or at work, about 5% of each day is spent in transit (table 1). Transportation environments pose unique problems with regard to air quality. In most, air exchange rates are high, but the ratio of the number of occupants to air volume is much higher than in other environments. For example, commercial jets are designed to have several air exchanges per hour. In an automobile with the windows open or the ventilation fan operating, the number of air exchanges can range from 5 to 50 per hour. The occupant-to-volume ratios are much higher than found in almost every indoor environment used by the general public. Thus, substantial exposure to airborne pollutants may be sustained in transportation environments.

The ventilation systems of commercial aircraft are designed to pressurize the cabin, cool electronic equipment, vent lavatory and galley areas, and provide conditioned air for passenger and crew comfort (30). The air is drawn through compressors, conditioned by mixing with ambient air, and delivered through overhead systems. The air exchange on airplanes is usually quite high, although some aircraft, such as the new Boeing 767, are configured to recirculate 50% of the air. At full passenger load, only 7 cubic feet per minute (cfm) of fresh air per person would be supplied, only slightly greater than the minimum recommended in the current ventilation guidelines of the American Society of Heating, Refrigerating, and Air Conditioning Engineers.

Important and occasionally unique exposures to pollutants can be sustained in special environments, although little time may be spent in these locations on average. For example, sports arenas can be heavily contaminated with tobacco smoke (31), and motorized ice cleaning equipment can increase levels of CO and NO_2 in ice skating rinks (32). Exposure to volatile organic compounds (VOC) can take place in dry cleaning establishments and fabric stores (33). Camping lanterns and cooking stoves are potent sources of NO_2 and CO (34). In fact, fatalities have occurred to campers and explorers from CO poisoning (35, 36).

TABLE 2

TYPICAL SOURCES OF INDOOR AIR POLLUTION IN THE HOME, OFFICE, AND TRANSPORTATION ENVIRONMENT

Environment	*Source and Pollutants*
Home	Tobacco smoking: respirable particles, CO, VOC*
	Gas stoves: NO_2, CO
	Woodstoves and fireplaces: respirable particles, CO, PAH†
	Building materials: formaldehyde, radon
	Earth underlying the home: radon
	Furnishings and household products: VOC, formaldehyde
	Gas-fueled space heaters: NO_2, CO
	Kerosene-fueled space heaters: NO_2, CO, SO_2
	Insulation: asbestos
	Moist materials and surfaces: biological agents
Office	Tobacco smoking: respirable particles, CO, VOC
	Building materials: VOC, formaldehyde
	Furnishings: VOC, formaldehyde
	Copying machines: VOC
	Air conditioning systems: biological agents, vehicle exhaust with combustion emissions containing particles, CO, and NO_2
Transportation	Tobacco smoking: respirable particles, CO, VOC
	Ambient air: ozone in jet aircraft, CO and lead in automobiles
	Auto air conditioners: biological agents

* Volatile organic compounds.
† Polycyclic aromatic hydrocarbons.

Sources of Indoor Air Pollution

Numerous sources of airborne contaminants have been identified in indoor environments (tables 2 and 3). The pollutant sources found in home, office, and transportation environments are listed in table 2. In table 3, we describe the sources of the principal pollutants and typical concentrations. Unvented combustion, evaporation of solvents, grinding, and abrasion can produce gaseous and particulate pollution indoors. Radon and its decay products accumulate indoors from soil, water, and building materials. Biological sources include growth of molds, fungi, and bacteria, and insects and pets.

In the home, the principal combustion sources are tobacco smoking, gas cooking stoves, and unvented kerosene heaters. Wood burning in stoves and fireplaces may also affect indoor air quality. Formaldehyde may be released from urea formaldehyde foam insulation (UFFI),

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TABLE 3
THE PRINCIPAL INDOOR POLLUTANTS, THEIR SOURCES AND TYPICAL CONCENTRATIONS

Pollutant	Typical Sources	Pollutant Concentrations	Relevant Standards	Comments
Respirable particles	Tobacco smoke, unvented kerosene heaters, wood and coal stoves, fireplaces, outside air, attached facilities, occupant activities	> 500 μg/m³ bars, meetings, waiting rooms with smoking 100 to 500 μg/m³ typical for smoking sections of [illegible] 10 to 100 μg/m³ typical [illegible] homes 1,000 μg/m³ with burning [illegible] or fireplaces	265 μg/m³ EPA 24-h standard ambient air 75 μg/m³ EPA annual standard ambient air 150 μg/m³ Japanese indoor standard	Current EPA standards are for total, and not only respirable suspended particles
NO, NO_2	Gas ranges and pilot lights, unvented kerosene and gas space heaters, gasoline engines, some gas floor furnaces, outside air	25 to 75 ppb typical range for homes with gas stoves 100 to 500 ppb peak values kitchens with gas stoves or kerosene gas heaters	160 ppb 1-h maximum, WHO guideline 50 ppb annual average EPA ambient standard	No current EPA short-term standard
CO	Gas ranges and pilot lights, unvented kerosene and gas space heaters, tobacco smoke, back drafting of water heater or furnace or woodstove, gasoline engines, camping lanterns and stoves Attached garages, street level intake vents, hockey rinks	> 50 ppm when oven used for heating > 50 ppm attached garages, air [illegible] 2 to 15 ppm cooking with gas stove 2 to 10 ppm heavy smoking in homes, bars, and other locations	35 ppm EPA 1-h standard 9 ppm EPA 8-h standard	
CO_2	People, unvented kerosene and gas space heaters, tobacco smoke, outside air	320 to 400 ppm outdoor air 2,000 to 5,000 ppm crowded indoor environment, inadequate ventilation	1,000 ppm Japanese indoor air standard	CO_2 concentrations below 1,000 ppm usually indicate adequate fresh air supply for buildings.
Infectious, allergenic, irritating biological materials	Dust mites and cockroaches, animal dander, bacteria, fungi, viruses, pollens	Few systematic measurements of spores, bacteria, and viruses indoors Homes with mold problem, offices with water damage: > 1,000 cfu/m³* Homes and offices without obvious problem: 800 ± 200 cfu/m³	None	Interpretations of a level depend on the specific agent; cfu/m is only an indicator
Formaldehyde	Urea formaldehyde foam insulation (UFFI), glues, fiberboard, pressed board, plywood, particle board; carpet backing and fabrics	0.1 to 0.6 ppm homes with UFFI 0.5 ppm average in mobile homes > 1 ppm in a few homes and mobile homes	0.2 to 0.6 ppm adopted by several states 0.1 ppm Sweden, new homes 0.7 ppm Sweden, maximum in old buildings 3 ppm U.S. OSHA 8-h time-weighted average	Formaldehyde concentrations in homes with UFFI decline by 50% every 2 to 3 yr.
Radon and radon daughters	Ground beneath a home, domestic water, and some utility natural gas	1.5 pCi/l estimated average in U.S. homes > 8 pCi/l in 3% to 5% homes	8 pCi/l NCRP action level 4 pCi/l EPA limit for uranium processing site homes 2 pCi/l ASHRAE guidelines 5 pCi/l Sweden, maximum, existing buildings 3 pCi/l Sweden, maximum, new buildings	Radon or radon daughters can be measured. Standards are for radon. Lung cancer risk results from radon daughters.
Volatile organic compounds: benzene, styrene, tetrachloroethylene; dichlorobenzene; methylene chloride; chloroform	Outgassing from water, plasticizers, solvents, paints, cleaning compounds, mothballs, resins, glues, gasoline, oils, combustion, art materials, photocopiers, personal care products	Typical indoor concentrations of selected compounds: benzene—15 μg/m³; 1,1,1 trichloroethylene—20 μg/m³; chloroform—2 μg/m³; tetrachloroethylene—5 μm/m³; styrene—2 μg/m³; m,p-dichlorobenzene—4 μg/m³; m,p-xylene—16 μg/m³	No indoor standards for nonoccupational settings	EPA Carcinogenic Assessment Group potency factors available for many of the volatile organics.
Semivolatile organics: Chlorinated hydrocarbons, DDT, heptachlor, chlordane	Pesticides, transformer fluids, termicides, combustion of wood, tobacco, kerosene, and charcoal; wood preservatives, fungicides	Only limited data available	No indoor standards	
Semivolatile organics: polycyclic compounds, benzo(a)pyrene, polychlorinated biphenols	Herbicides, insecticides			
Asbestos	Insulation on building structural components; asbestos plaster around pipes and furnaces; tiles	No systematic measurements to determine typical fiber concentrations. > 1,000 ng/m³ when friable asbestos	2 fibers/cc OSHA 8-h time-weighted average	EPA and state attention has been on schools and office buildings. Domestic problems not evaluated

* cfu/m³ = colony-forming units/m³.

from furnishings, and from household products. Volatile organic compounds have numerous sources within the home including tobacco smoking and household products. Radon, emitted by the earth under a home, can enter through cracks in the foundation, crawl spaces, sump holes, and other portals. Building materials, water, and utility natural gas may also be sources of radon.

In offices, tobacco smoking is an important source of respirable particulates. Formaldehyde and other VOC may be given off by building materials, furnishings, paints, waxes, supplies, and clean-

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ing solvents. Biological agents, which have proliferated on moist surfaces, may be dispersed by the office heating and cooling systems. Many of these same sources of air contamination are present in transportation environments.

Personal Exposure to Air Pollutants

Measurement of personal exposures to pollutants confirms the contributions of these indoor sources to total pollutant exposures (37). Direct personal monitoring has become possible with the development of passive sampling equipment and lightweight portable pump systems (38, 39). By combining personal sampling or fixed-location sampling with time-activity information, the relative contributions of various locations and sources to personal exposures can be estimated (37). Studies using these techniques have established the importance of indoor sources for exposure to respirable particulates, CO and NO_2.

For example, Spengler and associates (40) evaluated sources of variation in personal exposures to respirable particles among residents of 2 semirural communities in Tennessee. The ambient concentrations and personal exposures were uncorrelated, but the concentration of respirable particulates in the homes explained more than 60% of the variation in personal exposures. Reported tobacco smoke exposure alone accounted for less than 15% of the variability.

Quackenboss and colleagues (41) reported similar results from a study of personal NO_2 exposures of 35 adults and children living in the vicinity of an agricultural community in central Wisconsin. In this population more than 80% of the variance in week-long personal exposure to NO_2 was accounted for by variation in bedroom concentrations. In contrast, the studies of personal exposure have indicated the predominance of outdoor sources for some pollutants, e.g., O_3 (37).

Health Effects of Indoor Air Pollution

Introduction

We have described the sources of indoor air pollution and the principal pollutants that may be found in specific indoor environments. In this section, we address the health effects associated with these pollutants as well as information on the concentrations of the pollutants in indoor air. In discussing their health effects, we have broadly grouped the pollutants by their sources: combustion sources—tobacco smoke, NO_2, CO, and wood smoke; biological sources—infectious agents and allergens; and miscellaneous sources—radon and radon daughters, volatile organic compounds, and formaldehyde. The problem of building-related illnesses or "tight building syndrome," which cannot be linked to specific agents, is described separately. We do not review the hazards, primarily nonrespiratory, of exposure to pesticides. The National Research Council (42) has recently addressed the nonoccupational health risks of asbestiform fibers, and we do not cover this exposure. Finally, we do not consider the effects of pollutants generated by outdoor sources that penetrate indoors nor exposures in the work environment that are associated with well-recognized forms of occupational lung disease.

Tobacco Smoke

Introduction. Extensive toxicologic, experimental, and epidemiologic data, largely collected since the 1950s, have established that active cigarette smoking is a major preventable cause of morbidity and mortality (43). Involuntary exposure to tobacco smoke has only recently been investigated as a risk factor for disease in nonsmokers. Consequently, the evidence on involuntary smoking is more limited in scope than for active smoking, and controversy remains concerning certain associations of involuntary smoking with disease.

Nonsmokers inhale environmental tobacco smoke, the combination of the sidestream smoke that is released from the cigarette's burning end and the mainstream smoke exhaled by the active smoker (44). Comprehensive discussions of the chemistry of sidestream and of mainstream smoke are included in the 1979, 1984, and 1986 reports of the Surgeon General (43, 45, 46), in the 1981 report of the National Research Council on indoor air pollution (15), and in the 1986 report of the National Research Council on environmental tobacco smoke (47).

The exposures of involuntary and active smoking differ quantitatively and, to some extent, qualitatively (15, 45–48). Because of the lower temperature in the burning cone of the smoldering cigarette, most partial pyrolysis products are enriched in sidestream as compared to mainstream smoke. Consequently, sidestream smoke has higher concentrations of some toxic and carcinogenic substances than mainstream smoke; however, dilution by room air markedly reduces the concentrations inhaled by the involuntary smoker in comparison to those inhaled by the active smoker. Nevertheless, involuntary smoking is accompanied by exposure to many of the toxic agents generated by tobacco combustion (15, 45–48). The intake of tobacco smoke components by nonsmokers has been confirmed by studies using biological markers such as nicotine and its metabolite, cotinine. Thus, it is biologically plausible to hypothesize that exposure to environmental tobacco smoke is a risk factor for disease in nonsmokers. Active smokers must necessarily have greater exposure to environmental tobacco smoke than nonsmokers, but the consequences of smokers' active and passive exposures cannot be separately evaluated.

To date, research on passive smoking has focused on respiratory effects, although recent investigations have examined associations with diverse health effects including nonrespiratory cancers, ischemic heart disease, age at menopause (49), sudden infant death syndrome (50), and birth weight (51, 52). This review will emphasize the respiratory effects of involuntary smoking. Because the literature on passive smoking has been reviewed in this journal (53), in the 1984 and the 1986 reports of the Surgeon General (45, 46), and by the National Research Council (47), we will focus on the newer studies and the converging evidence for some effects of involuntary smoking. Symposia (18, 54, 55) and a monograph by Shephard (56) have also addressed the adverse health effects of involuntary smoking. Other reviews on selected aspects of the health effects of involuntary smoking have also been published (57–61).

Exposure to Environmental Tobacco Smoke. Tobacco smoke is a complex mixture of gases and particles that contains myriad chemical species (43, 45). Not surprisingly, tobacco smoking in indoor environments increases levels of respirable particulates, nicotine, polycyclic aromatic hydrocarbons, CO, acrolein, NO_2, and many other substances. The extent of the increase varies with the number of smokers, the intensity of their smoking, the ventilation rate of the indoor space, and the use of air cleaning devices. Several cigarette smoke components have been measured in indoor environments as markers of the contribution of tobacco combustion to indoor air pollution. Particulates have been measured most often: sidestream and mainstream smoke both

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contain high concentrations of particles in the respirable size range (46, 47). However, surveys of indoor air quality based on measurement of total suspended particulate concentrations will not readily identify the excess mass indoors from environmental tobacco smoke. Studies of levels of environmental tobacco smoke components have been conducted largely in various public buildings; fewer studies have been conducted in the home and office environments (46, 47).

The contribution of smoking in the home to indoor air pollution has been demonstrated by studies involving personal monitoring and monitoring of homes for respirable particulates. Spengler and associates (62) monitored 80 homes for respirable particulate concentrations over several years and found that a smoker of 1 pack of cigarettes daily contributed about 20 μg/m³ to 24-h indoor particle concentrations. In homes with 2 or more heavy smokers, this study showed that the 24-h National Ambient Air Quality Standard of 260 μg/m³ for total suspended particulates could be exceeded. Because cigarettes are not smoked uniformly over the day, higher peak concentrations must occur when cigarettes are actually smoked. Therefore, short-term total suspended particulate concentrations of 500 to 1,000 μg/m³ are likely at the time when cigarettes are actually smoked. The dramatic effect of smoking in the home is shown in figure 2, which summarizes 24-h samples of respirable suspended particulates in residences (63). The variation in the excess indoor concentrations among residences was related to the number of smokers and the air exchange rates. Home 11 had an average air exchange rate of only 0.44 per hour.

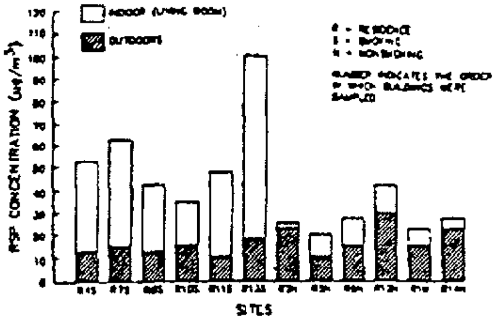

Fig. 2. Average 24-h respirable suspended particulate (RSP) concentrations (μg/m³) outdoors and indoors in homes with and without smokers. (Redrawn with permission from reference 63.)

Spengler and associates (40) measured the personal exposures to respirable particulates sustained by nonsmoking adults in 2 rural Tennessee communities. The mean 24-h exposures were substantially higher for those exposed to smoke at home.

Levels of other constituents of tobacco smoke have not been well characterized in homes. In the studies of Moschandreas and associates (63), only homes with smokers had iron, arsenic, and cadmium levels indoors that exceeded outdoor concentrations. Ambient cadmium ranged between 1 and 2.5 ng/m³ while the indoor home average concentrations ranged between 2 and 5 ng/m³ in the homes with heavy smoking. Under usual circumstances of smoking, the average emission rate of CO, 50 mg per cigarette, will not increase concentrations in a residence to the standards set for outdoor air (64).

More extensive information is available on levels of environmental tobacco smoke in various public buildings. Monitoring in locations where smoking may be intense, such as bars and restaurants, has generally shown elevations of particulates and other markers of smoke pollution where smoking is taking place (46, 47). For example, Repace and Lowrey (65) used a piezobalance to sample aerosols in restaurants, bars, and other locations. They found that respirable particulate levels ranged up to 700 μg/m³ and varied with the intensity of smoking. Similar data have been reported for the office environment, though the information is more limited (46, 47).

Transportation environments may also be affected by cigarette smoking. Contamination of air in trains, buses, automobiles, airplanes, and submarines has been documented (46, 47). For example, a recent National Research Council Report (30) on air quality in airliners summarized studies of tobacco smoke pollutants in commercial aircraft. During a single flight, the NO_2 concentration varied with the number of passengers with a lighted cigarette. In another study, respirable particulates in the smoking section were measured at concentrations 5-fold or greater than in the nonsmoking section. Peaks as high as 1,000 μg/m³ were measured in the smoking section.

Markers of Exposure. Biological markers of tobacco smoke exposure have been used to describe the prevalence and the dosimetry of involuntary smoking. In both active and involuntary smokers, the detection of various smoke components or their metabolites in body fluids or alveolar air provides evidence of exposure, and levels of these markers can be used to gauge the intensity of exposure to tobacco smoke. The risks of involuntary smoking have also been estimated by comparing levels of biological markers in active and involuntary smokers.

At present, the most sensitive and specific markers for tobacco smoke exposure are nicotine and its metabolite, cotinine (47, 66). Neither nicotine or cotinine are present in body fluids without exposure to tobacco smoke. Because the circulating half-life of nicotine is generally less than 2 h (67), nicotine concentrations in body fluids reflect recent exposures. In contrast, cotinine has a half-life in the blood or plasma of active smokers that ranges from less than 10 h to about 40 h (68, 69). The half-life of cotinine tends to be longer in nonsmokers than in smokers (68). Hence, cotinine levels provide information about more chronic exposure to tobacco smoke in both active and involuntary smokers. Thiocyanate concentration in body fluids, concentration of CO in expired air, and carboxyhemoglobin level distinguish active smokers from nonsmokers, but these measures are not as accurate as cotinine for assessing involuntary exposure to tobacco smoke (66, 70, 71).

Recent reports described cotinine levels and their relationship to exposure in adult nonsmokers and in children (table 4). In adult nonsmokers, exposures at home, in the workplace, and in other settings determined cotinine concentrations in urine and saliva. The cotinine levels associated with involuntary smoking ranged from less than 1% to about 8% of cotinine levels measured in active smokers. As would be anticipated, smoking by parents was the predominant determinant of the cotinine levels in children. Greenberg and colleagues (75) found significantly higher concentrations of cotinine in the urine and saliva of infants exposed to cigarette smoke in their homes in comparison with unexposed controls. Urinary cotinine levels in the infants increased with the number of cig-

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TABLE 4

SELECTED STUDIES OF COTININE LEVELS IN NONSMOKERS

Population	Findings
100 adult patients attending clinics in London (72)	Cotinine levels parallel self-reported exposure. In nonsmoking, mean = 1.5 ng/ml in saliva; in active smokers, mean = 309.9 ng/ml.
151 adult males attending a clinic in London and 70 subjects from Oxford (73, 74)	Urinary cotinine level increased with reported duration of smoke exposure. In nonexposed nonsmokers, median level = 2.0 ng/ml; exposed nonsmokers, median = 6.0 ng/ml; active cigarette smokers, median = 1,645 ng/ml. Smoking by wife increased cotinine concentrations 3-fold in nonsmoking men.
51 infants attending clinics in North Carolina (75).	In nonexposed, median urinary level = 4 ng/mg creatinine; exposed, median = 351 ng/mg creatinine. Salivary creatinine levels higher in exposed.
472 nonsmoking adults in Japan (76).	Exposure at home and at work independently increased urinary cotinine level. In nonsmokers, median = 680 ng/mg creatinine; active smokers, median = 8,570 ng/mg creatinine.
569 schoolchildren, 11 to 16 yr of age, in Bristol, England (77).	Salivary cotinine level increased with the number of smoking parents. If neither parent smoked, median = 0.20 ng/ml; if both smoked, median = 2.70 ng/ml.
38 children, 3 to 15 months of age, attending a child center in North Carolina (78).	Serum cotinine level increased with the number of smokers in the home. In children without household exposure, mean = 1.0 ng/ml; exposed, mean = 4.1 ng/ml.
839 children and adults in a population sample in New Mexico (79).	Salivary cotinine level increased with the number of smokers in the home. If no smokers in the home, median level was zero. If 1 or more smokers, median was greater than zero and increased with the number of smokers.

arettes smoked during the previous 24 h by the mother. The findings were similar in another study of infants that was based on serum cotinine levels (78). Luck and Nau (80) have shown that cotinine and nicotine levels measured in infants breastfed by smoking mothers reflect both the doses received from the breast milk and from involuntary smoking. In a study of schoolchildren in England, salivary cotinine levels rose with the number of smoking parents in the home (77). Similar data were recently reported from a large population-based sample in New Mexico (79).

The results of some investigations based on other markers of exposure have been similar. For example, Poulton and associates (81) reported that serum thiocyanate levels were significantly higher in children living with smokers. In fact, levels of thiocyanate are increased in umbilical cord blood if the mother lives with smokers (82). Nicotine levels in adults vary with report of recent exposure, and in several English studies all nonsmokers had measurable concentrations of nicotine in body fluids (66, 83, 84).

The results of these studies using biological markers have important implications for research on involuntary smoking. The studies provide ample evidence that involuntary exposure leads to absorption, circulation, and excretion of tobacco smoke components, and the studies confirm the high prevalence of involuntary smoking, as ascertained by questionnaire (85). The results further suggest that questionnaire methods for assessing recent exposure have some validity. These studies also demonstrate that saliva and urine samples can be readily obtained from large populations; thus, cotinine levels in body fluids could be used as a marker of exposure in large-scale epidemiologic research on involuntary smoking. However, further investigation is needed to define the relationship between inhaled nicotine and cotinine levels in body fluids, the extent to which cotinine levels index doses of other tobacco smoke components, and the range and determinants of cotinine half-life in nonsmokers. Further, a biological marker for cumulative exposure, which would facilitate investigation of chronic diseases, has not been identified.

Comparisons of levels of biological markers in smokers and nonsmokers have been made in order to estimate the relative intensities of active and involuntary smoking. However, a simple proportionality cannot be assumed between the ratio of the levels of markers in passive and active smokers and the relative doses of all tobacco smoke components. Nonetheless, several investigators have attempted to characterize involuntary smoking in terms of active smoking. For example, Foliart and coworkers (86) measured urinary excretion of nicotine in flight attendants during an 8-h flight and estimated that the average exposure was 0.12 to 0.25 mg of nicotine. Russell and colleagues (87) compared nicotine levels in nonsmokers exposed to tobacco smoke with levels achieved after infusion of known doses of nicotine. On the basis of this comparison, the investigators estimated that the average rate of nicotine absorption was 0.23 mg per hour in a smoky tavern, 0.36 mg per hour in an unventilated smoke-filled room, and 0.014 mg per hour from average daily exposure. In active smokers, the first cigarette of the day resulted in absorption of 1.4 mg of nicotine.

Nonmalignant Respiratory Effects. The 1983 "State of the Art" review on involuntary smoking (53) and the 1984 and the 1986 reports of the Surgeon General (45, 46) provide comprehensive summaries of the literature on respiratory effects of involuntary smoking other than lung cancer. These publications have concurred in concluding that for children passive smoking increases the occurrence of lower respiratory illness, particularly early in life, and increases the frequency of chronic respiratory symptoms. On the basis of primarily cross-sectional data, the 1984 report of the Surgeon General (45) also concluded that the children of smoking parents in comparison with those of nonsmokers had small reductions of lung function, but the long-term consequences of these changes were regarded as unknown. In the 2 yr between the 1984 and the 1986 reports, sufficient longitudinal evidence accumulated to support the conclusion in the 1986 report (46) that involuntary smoking reduces the rate of lung function growth during childhood. Only limited data pertaining to adults have been available, and definitive conclusions have not been made for adult populations.

The more recent data on children have generally supported the conclusions of the earlier review in this journal (53) and of the Surgeon General's reports. With regard to respiratory illness in infants, Pedreira and colleagues (88) prospectively monitored the incidence of lower respiratory illness in 1,144 infants followed in a pediatric practice. Office visits

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for tracheitis and bronchitis were significantly more common for infants exposed to tobacco smoke at home. The effects of prenatal smoke exposure could not be separated from those of postnatal exposure in previous studies of lower respiratory illness. However, relevant data have been published from 2 populations. A prospective study in China of 1,058 infants of nonsmoking mothers demonstrated that paternal smoking increased the rate of hospitalization for respiratory illness during the first 18 months of life (89). A British cohort study suggested independent effects of prenatal and postnatal exposure on lower respiratory illness experience in early life (90).

Data from 2 large cross-sectional investigations demonstrated an association between parental smoking habits and lower respiratory illness before 2 yr of age (91, 92). Ware and associates (92) analyzed questionnaire information from 10,106 children, 6 to 9 yr of age at enrollment, who were participating in the Harvard Air Pollution Health Study in 6 U.S. cities. Smoking by both the mother and the father was associated with a higher frequency of reported physician-diagnosed respiratory illness before 2 yr of age. The relative odds for this illness variable increased progressively with the usual number of cigarettes smoked daily by the mother at the time of interview. In a prevalence survey of 1,355 Iowa children 6 to 12 yr of age, parental smoking significantly increased the risk of hospitalization for a chest illness before 2 yr of age (91). Although recall of past illnesses may be inaccurate (93), bias in reporting that depends upon parental smoking habits is unlikely.

Two recent studies did not show effects of involuntary smoking on respiratory illnesses in children. Gardner and colleagues (94) monitored 131 infants during the first year of life for viral infections by serology, cultures, and clinical examinations. Neither specific infections nor illnesses were associated with parental smoking habits. The study population was small, however, and did not have sufficient statistical power to examine effects in the range of interest. In a study based on data from a health maintenance organization, Vogt (95) found that household smoking characteristics did not influence use of outpatient care services for respiratory illness by children.

New studies have showed that children exposed to cigarette smoke in their homes are also at increased risk for middle ear disease. Both acute otitis media (96) and persistent middle ear effusions (97-99) have been associated with involuntary smoking.

The more recent studies continue to indicate increased respiratory symptoms in the children of smokers. In the Harvard Air Pollution Health Study, smoking by parents increased the frequency of cough and wheeze in their children by up to about 30% (92). Analysis of data from 3,482 nonsmoking children, collected in 1962 to 1965 in Tecumseh, Michigan, also indicated more frequent respiratory symptoms in the children of smokers (100, 101). Charlton (102) conducted a survey on cigarette smoking that included 15,709 English children 8 to 19 yr of age. In the nonsmoking children, the prevalence of frequent cough was significantly higher if either the father or the mother smoked, in comparison with the prevalence when neither parent smoked.

The findings of the newer studies are inconsistent on the relationship between passive smoking and wheezing and asthma. McConnochie and Roghmann (103) assessed predictors of wheeze in a retrospective cohort study of children who had mild bronchiolitis in infancy and of control children without illness. At a mean age of 8.3 yr, current exposure to tobacco smoke at home was a significant predictor of wheeze (odds ratio = 1.9, $p = 0.05$). Further analysis of data from the control children showed that maternal smoking significantly increased the prevalence of wheezing on follow-up in children from families with a history of respiratory allergy (104). In the study of children in Tecumseh, Michigan, parental smoking was associated with a higher prevalence of asthma at the initial examination and with a doubling of the risk for developing asthma during the 15-yr follow-up period (100, 101). Murray and Morrison (105) evaluated 94 asthmatic children 7 to 17 yr of age. Level of lung function, symptom frequency, and responsiveness to inhaled histamine were adversely affected by maternal smoking.

In contrast, Tashkin and associates (106) examined cross-sectional data from children 7 to 17 yr of age in the Los Angeles area and found no association between the smoking characteristics within the households and the prevalence of respiratory symptoms or asthma. In a prospective cohort study in New Zealand, parental smoking habits were not found to affect the incidence of asthma during the first 6 yr of life (107). In 1980, Weiss and associates (108) reported the results of a cross-sectional survey of respiratory symptoms in 650 children in Massachusetts. The prevalence of persistent wheeze, the most common symptom, increased significantly with the number of smoking parents but was unrelated to smoking by the children themselves. These investigators subsequently used cold air challenge to assess airways responsiveness in a sample of these children and found that airways reactivity was not related to maternal smoking history (109).

New studies have further documented the adverse effect of parental smoking on children's lung function, and longitudinal evidence on the consequences of passive smoking during lung growth and development was published. In the study of children in Tecumseh, Michigan, parental smoking was associated with reduced lung function, as assessed by spirometry (100, 101). The magnitude of effect varied with age, sex, and the index of exposure to parental smoking. In the survey in Los Angeles, maternal smoking was associated with average reductions of 3 to 8% for spirometric parameters in male subjects (106). The effects of paternal smoking were largest in boys less than 12 yr of age and were variable in girls. Ekwo and coworkers (91) found significantly greater response to inhaled bronchodilator in the children of cigarette smokers. In the Harvard Air Pollution Health Study, the FEV_1 of children whose mothers smoked at the time of spirometry was reduced by slightly less than 1% of predicted FEV_1 reduction (92). In contrast, Hosein and Corey (110) studied 1,357 children and did not find an effect of home exposure to tobacco smoke on FEV_1 level. Lebowitz and colleagues (111) also did not find effects of parental smoking, but only 271 children were included in the study population.

Based on cross-sectional data from children in East Boston, Massachusetts, Tager and associates (112) reported in 1979 that the level of FEF_{25-75} declined with the number of smoking parents in the household. In 1983, these investigators provided the results obtained with follow-up of these children over a 7-year period (113). Using a multivariate technique, Tager and associates showed that both maternal smoking and active smoking by the child reduced the growth rate of the FEV_1. The statistical model predicted effects of maternal smoking that are of a physiologically important magnitude. Lifelong exposure of a child to a smoking mother was estimated to reduce growth of the FEV_1 by 10.7, 9.5,

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and 7.0% after 1, 2, and 5 yr of follow-up, respectively.

Recent longitudinal data from the Harvard Air Pollution Health Study also showed reduced growth of the FEV_1 in children whose mothers smoked cigarettes (114). The growth rate of the FEV_1 from ages 6 through 10 yr was calculated for 7,834 white children. Although all representations of exposure to parental smoking were associated with reduced growth rate of the FEV_1, only the level of maternal smoking in packs per day attained statistical significance at $p < 0.05$. From ages 6 through 10 yr, the statistical model estimated that FEV_1 growth rate is reduced by 0.17% per pack of cigarettes smoked daily by the mother. This effect was somewhat smaller than that reported earlier by Tager and associates (113), although if extrapolated to age 20 yr, a cumulative effect of 2.8% is predicted. The 2 sets of data were also analyzed with noncomparable techniques, and the study populations may have differing levels of exposure to passive smoking.

Burchfiel (100, 101) examined the effects of parental smoking on 15-yr lung function change of subjects in the Tecumseh study, first examined at ages 10 through 19 yr. In the female subjects who remained nonsmokers across the follow-up period, parental smoking was not associated with lung function change. In nonsmoking males, parental smoking reduced the growth of the FEV_1, FVC, and $\dot{V}max_{50}$, although the sample size was limited and the effects were not statistically significant. For the FEV_1 in males, the analysis estimated 7.4% and 9.4% reductions in 15-yr growth associated with 1 or 2 smoking parents, respectively.

Some new information has become available for adults since the previous reviews, which cited data from only 4 epidemiologic studies. The ratio of hydroxyproline to creatinine in urine was used by Japanese investigators as a marker of lung injury (115). In women passively exposed to cigarette smoke, this ratio increased with the extent of daily exposure. However, in a study in Germany, the hydroxyproline to creatinine ratio in nonsmokers did not vary with passive smoke exposure (116). Moreover, Read and Thornton (117) reported that in experimental studies with rats, the hydroxyproline to creatinine ratio actually decreased with increasing exposure to smoke. They also reported that in humans both hydroxyproline and creatinine individually increased with increased nicotine absorption from active smoking in males but not in females (117). The ratio of the two, however, was not associated with increased nicotine excretion in either sex.

The results of several of the more recent epidemiologic studies indicate possible chronic effects of passive smoking on lung function in adults. The results of an investigation of 163 nonsmoking women in the Netherlands suggested adverse effects of tobacco smoke exposure in the home (118, 119). Cross-sectional analysis of spirometric data collected in 1982 showed reductions of most parameters in association with tobacco smoke exposure in the home, although the effect was significant only for flows at higher lung volumes. In a sample of the women, domestic tobacco smoke exposure was not associated with longitudinal decline of lung function during the period 1965 to 1982. In baseline data for a cohort study in Scotland, respiratory symptoms tended to be more prevalent in nonsmokers living with smokers in comparison to nonsmokers living with nonsmokers (120).

Other studies have not indicated chronic effects of passive tobacco smoke exposure on adult nonsmokers. Jones and associates (121) conducted a case-control study of 20- to 39-yr-old nonsmoking women in the Tecumseh Community Health Study cohort. Subjects from the highest and lowest quartiles of the lung function distribution had comparable exposure to smokers in the home. Kentner and colleagues (122) in a study conducted in Germany examined the effects of passive and active smoking in 1,351 white collar workers. Self-reported exposure to environmental tobacco smoke at home and at work was not associated with reduction of lung function, as assessed by spirometry. In a small case-control study, marriage to a smoker was not associated with excess risk for chronic bronchitis (123).

New experimental and epidemiologic studies have not consistently shown acute effects of passive smoking on lung function level in asthmatic and nonasthmatic children and adults. As described above, Murray and Morrison (105) found lower ventilatory function in asthmatic children with smoking mothers. In a population sample in Tucson, Arizona, Lebowitz (124, 125) examined the relationship between passive smoking and daily symptom occurrence and daily level of peak flow. Statistically significant effects of tobacco smoke exposure were not found for either outcome in the 229 children and adults. In an experimental study, 1-h chamber exposure of young asthmatics to cigarette smoke did not reduce expiratory flow rates and was, in fact, followed by a small decrease in nonspecific airways reactivity (126).

The accumulating evidence since previous reviews continues to demonstrate adverse effects of passive smoking on the lungs of children. Data from large populations showed significant effects on lung function level and symptom occurrence (91, 92, 102, 106). Results from follow-up of the East Boston, the Harvard, and the Tecumseh study cohorts (100, 101, 113, 114) suggested that the effects on lung function should not be dismissed as clinically insignificant.

Important research questions pertaining to passive smoking and the child's lung remain unanswered, however (46, 127). The mechanisms of injury have not been established, and the relative importance of exposures *in utero*, during infancy, and later in childhood has not been examined. Nevertheless, the available evidence of adverse effects does provide sufficient rationale for intervention. In contrast to the evidence for children, the data on adults are more variable and do not yet permit conclusive statements concerning passive smoking during adulthood and reductions of lung function and increased respiratory symptom occurrence.

Lung Cancer. In 1981, reports were published from Japan (128) and from Greece (129) that indicated increased lung cancer risk in nonsmoking women married to cigarette smokers. Subsequently, this controversial association has been examined in investigations conducted in the United States, Scotland, Japan, and Hong Kong. The association of involuntary smoking with lung cancer derives biological plausibility from the presence of carcinogens in sidestream smoke and the lack of a documented threshold dose for respiratory carcinogenesis in active smokers (130). Further, mutagenic activity can be found in the urine of nonsmokers after passive exposure to tobacco smoke (131, 132).

Time trends of lung cancer mortality in nonsmokers have been examined with the rationale that temporally increasing exposure to environmental tobacco smoke should be paralleled by increasing mortality rates. Enstrom (133) calculated nationwide lung cancer mortality rates for 1914 to 1968 and concluded that a real increase had occurred among nonsmoking males after 1935. However, occupational and environmental exposures

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TABLE 5

COHORT AND CASE-CONTROL STUDIES OF PASSIVE EXPOSURE TO TOBACCO SMOKE AND LUNG CANCER

Study	Findings	Comment
Prospective cohort study in Japan of 91,540 nonsmoking females, 1966–1981 (136).	Age-occupation adjusted SMR,* by husband smoking: Nonsmokers—1.00 Ex-smokers—1.36 < 20/day—1.45 ≥ 20/day—1.91	Trend statistically significant. All histologies
Case-control study in Greece of 40 nonsmoking female cases, 149 controls, 1978–1980 (129)	Odds ratios by husband smoking: Nonsmokers—1.0 Ex-smokers—1.8 Current smokers < 20/day—2.4 > 20/day—3.4	Trend statistically significant. Histologies other than adenocarcinoma and bronchioloalveolar carcinoma.
Prospective cohort study in the U.S. of 176,139 nonsmoking females, 1960–1972 (134).	Age-adjusted SMR, by husband smoking: Nonsmokers—1.00 Current smokers < 20/day—1.27 ≥ 20/day—1.10	All histologies. Effect of husband smoking not significant
Case-control study in Hong Kong of 84 female cases and 139 controls, 1976–1977 (137, 138).	Crude odds ratio of 0.75 associated with smoking spouse	All histologies. Two reports are inconsistent on the exposure variable.
Case-control study in the U.S. with 22 female and 8 male nonsmoking cases, 133 female and 180 male controls (139).	Odds ratios by spouse smoking: Nonsmokers—1.00 < 40 pack years—1.48 ≥ 41 pack years—3.11	Significant increase for > 41 pack years. Bronchioloalveolar carcinoma excluded.
Case-control study in the USA, 25 male and 53 female nonsmoking cases with matched controls, 1971–1980 (140).	Odds ratio not significantly increased for current exposure at home: Males—1.26 Females—0.92	All histologies. Findings negative for spouse smoking variable as well.
Prospective cohort study in Scotland of 8,128 males and females, 1972–1982 (120).	Age-adjusted mortality ratios for domestic exposure: Males—3.25 Females—1.00	Preliminary, small numbers of cases.
Case-control study in Hong Kong with 88 nonsmoking female cases, 1981–1982 (141, 142).	Odds ratio of 1.24 (p > 0.40) for combined home and workplace exposure. No association with cumulative hours of exposure.	All histologies.
Case-control study in the U.S. with 31 nonsmoking and 189 smoking female cases (143).	No significant effects of exposure from parents, spouse, or workplace in smokers and nonsmokers.	Adenocarcinoma and squamous cell carcinoma only.
Case-control study in the U.S. with 134 nonsmoking female cases (135).	Nonsignificant odds ratio of 1.22 if husband smoked. Significantly increased odds ratio of 2.11 if husband smoked 20 or more cigarettes daily at home. Significant trend with number of cigarettes smoked at home by the husband.	All histologies. Careful exclusion of smokers from the case group.
Case-control study in England with 15 male and 32 female nonsmoking cases, and 30 male and 96 female nonsmoking controls (123).	Overall odds ratio for spouse smoking of 1.1.	Hospital-based study.
Case-control study in Japan with 19 male and 94 female nonsmoking cases, and 110 male and 270 female nonsmoking controls (144).	For females, odds ratio of 1.5 if husband smoked; for males, odds ratio of 1.8 if wife smoked.	Clinical or radiologic diagnosis for 43%. All histologies.
Case-control study in Louisiana, Texas, and New Jersey with 99 nonsmoking cases and [illegible] controls (145).	Adjusted odds ratio for marriage to a smoking spouse was 1.5.	Nearly 100% histologic confirmation. All histologies.
Case-control study in New Mexico with 25 nonsmoking cases and 292 nonsmoking controls (146).	Adjusted odds ratio for marriage to a smoking spouse was 3.2. No effect in active smokers.	All histologies other than bronchioloalveolar carcinoma.

* Standardized mortality ratio.

other than environmental tobacco smoke could explain the apparent mortality rate increase in males. Garfinkel (134) did not identify similar trends in nonsmokers in the Dorn study of male U.S. veterans, 1954 to 1969, nor in the American Cancer Society's study of males and females, 1960 to 1972.

Epidemiologists have tested the association between lung cancer and involuntary smoking using conventional designs: the case-control and cohort studies. The results of both study designs may be affected by inaccurate assessment of exposure to environmental tobacco smoke, by inadequate information on personal smoking habits that leads to classification of smokers as nonsmokers, and by the misdiagnosis of a cancer at another site as primary cancer of the lung. For example, in the case-control study reported by Garfinkel and colleagues (135), 13% of cases originally diagnosed as lung cancer were reclassified to other sites after histological review and 40% of the cases initially classified as nonsmokers by chart review were found to be smokers on interview. The difficulty of accurately estimating exposures with questionnaires and descriptions of a spouse's smoking may partly explain the variable findings of the published studies. In fact, the validity and reliability of questionnaires on involuntary smoke exposure have yet to be comprehensively evaluated.

The evidence from the case-control and the cohort studies does not uniformly indicate increased lung cancer risk in persons exposed to environmental tobacco smoke, but most of the studies indicate increased risk in nonsmokers married to smokers (table 5). Hirayama

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(128) conducted a prospective cohort study of 91,540 nonsmoking women in Japan. Standardized mortality ratios for lung cancer increased significantly with the amount smoked by the husbands. The findings could not be explained by confounding factors and were unchanged when follow-up of the study group was extended (136). After its publication, this article received intensive scrutiny, and correspondence in the *British Medical Journal* raised concerns about statistical methodology, population selection, uncontrolled confounding, and the seemingly high relative risk; in his responses, Hirayama satisfactorily rebuffed most of these criticisms, although he could not eliminate the possibility of unreported smoking by women classified as nonsmokers (147). Based on the same cohort, Hirayama has also reported significantly elevated standardized mortality ratios for lung cancer of 2.1 and 2.3 in nonsmoking men with wives smoking 1 to 19 cigarettes and 20 or more cigarettes daily, respectively (136).

In 1981, Trichopoulos and colleagues (129) also reported increased lung cancer risk in nonsmoking women married to cigarette smokers. These investigators conducted a case-control study in Athens, Greece, that included cases with a diagnosis other than adenocarcinoma or bronchioalveolar carcinoma and controls selected at a hospital for orthopedic disorders. The findings were unchanged with expansion of the study population (148).

The results of other subsequently reported case-control studies have also demonstrated statistically significant associations between involuntary smoking and lung cancer (135, 144–146) (table 5). The findings from the more recent reports greatly strengthen the evidence from the earlier studies. Several of the newer studies included relatively large numbers of nonsmokers (135, 144, 145). Furthermore, in most of these studies, involuntary smoking was assessed in greater detail than in the earlier reports so that exposure-response relationships could be more fully examined.

The results of 2 other investigations have also been interpreted as showing an increased lung cancer risk associated with passive smoking, although both have methodologic limitations. In Germany, Knoth and colleagues (149) described a series of 59 female lung cancer cases of which 39 were in nonsmokers. Based on census data, the report by Knoth and colleagues projected that a much greater than expected proportion of these nonsmokers had lived in households with smokers. This report did not include an appropriate comparison series, however, and the suitability of substituting census data was not addressed by the authors. In another recent report, Gillis and associates (120) described the preliminary results of a cohort study of 16,171 males and females in western Scotland; domestic exposure to tobacco smoke increased the lung cancer risk for nonsmoking men but not for women. The report was based on only 16 cases of lung cancer in nonsmokers, however.

The results of other investigations indicate lesser or no effects of exposure to environmental tobacco smoke (table 5). In these studies, however, confidence limits for the relative risks associated with marriage to a smoker are wide and overlap with the confidence limits in the studies with significant results (47). Two separate case-control studies in Hong Kong, where lung cancer incidence rates in females are particularly high, did not indicate excess risk from passive smoking (137, 138, 141, 142). In the more recent of the 2 studies, the questionnaire comprehensively assessed cumulative exposure from home and workplace sources (141, 142). Lee and colleagues (123) reported a hospital-based case-control study in England. Although the investigators considered that their findings indicated little or no effect of involuntary smoking, the case series was small.

The results of the American Cancer Society's prospective cohort study of mortality in 176,139 nonsmoking women have also been construed by many as negative (134). However, the standardized mortality ratios for the nonsmoking women with husbands who smoked were greater than unity but not significantly greater. Repace (150) has suggested that the mortality ratios in the American Cancer Society cohort have been reduced by misclassification introduced by workplace exposures, a factor not considered in the original analyses. Recent and preliminary results from a nationwide case-control study also did not demonstrate increased lung cancer risk from domestic exposure to tobacco smoke (140). In another case-control study that was performed in Los Angeles, Wu and colleagues (143) did not find significantly increased risk for adenocarcinoma associated with involuntary smoking in smoking and nonsmoking women. These investigators estimated exposure from parental smoking, spouse smoking, and workplace sources. The relative risk for lung cancer was slightly, but not significantly, increased by exposure from spouse smoking and from smoking by coworkers.

At present, relatively few investigations provide data on the hypothesis that involuntary smoking is a risk factor for lung cancer. The extent of data contrasts with the more extensive literature cited in the 1964 Surgeon General's Report, which characterized active cigarette smoking as a cause of lung cancer (151). The variability of the data on involuntary smoking also contrasts with that on active smoking. However, most of the studies on involuntary smoking and lung cancer have small numbers of cases, and confidence intervals for the effect of involuntary smoking in the various studies would overlap. Variation in the results of the studies may also reflect random and nonrandom errors in the classification of exposure to environmental tobacco smoke. In fact, the problems of dose estimation seem more difficult for lung cancer than for other health effects of involuntary smoking. The relevant exposures may begin at birth and occur under a wide variety of circumstances. Thus, some inconsistency of the studies would be anticipated.

In spite of the variable epidemiologic evidence, environmental tobacco smoke has been recently characterized as a respiratory carcinogen. The International Agency for Research on Cancer of the World Health Organization (152) has concluded that "passive smoking gives rise to some risk of cancer." The agency supported this conclusion in its monograph on tobacco smoking by citing the characteristics of sidestream and mainstream smoke, the absorption of tobacco smoke materials during involuntary smoking, and the nature of dose-response relationships for carcinogenesis. Appropriately, the International Agency for Research on Cancer argued on the basis of biological plausibility rather than on the basis of epidemiologic evidence.

The National Research Council (47) and the U.S. Surgeon General (46) have also concluded that involuntary smoking increases the incidence of lung cancer in nonsmokers. In reaching this conclusion, the National Research Council (47) cited the biological plausibility of an association between environmental tobacco smoke exposure and lung cancer and the supporting epidemiologic evidence. This report carefully considered the sources of bias that may have affected the epidemiologic studies. Based on a

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pooled analysis of the epidemiologic data and adjustment for bias, the report's authors concluded that the best estimate for the excess risk of lung cancer in nonsmokers married to smokers was 25%. The 1986 report of the Surgeon General (46) characterized involuntary smoking as a cause of lung cancer in nonsmokers. This conclusion was based on the extensive information already available on the carcinogenicity of active smoking, on the qualitative similarities between environmental tobacco smoke and mainstream smoke, and on the epidemiologic data on involuntary smoking.

The extent of the lung cancer hazard associated with involuntary smoking in the United States remains uncertain, however (46, 153). The epidemiologic studies provide varying and imprecise measures of risk, and dosimetric aspects of involuntary smoking in the respiratory tract are not yet well understood. Risk estimation procedures have been used to describe the lung cancer risk associated with involuntary smoking, but assumptions and simplifications must be made in order to use this method. For example, Repace and Lowrey (154) have recently calculated that approximately 5,000 lung cancer deaths occur annually in U.S. nonsmokers as a result of involuntary smoking. The results of more refined risk estimation models should be forthcoming.

Other Cancers. Recent reports suggest that environmental tobacco smoke exposure may increase risk of cancer at sites other than the lung. One study found that in children, maternal exposure to environmental tobacco smoke during pregnancy was associated with increased risk of brain tumors (155), and in another study paternal but not maternal smoking increased the risk of childhood rhabdomyosarcoma (156). Such effects might arise from smoking-induced changes in germ cells of the parents or through transplacental exposure rather than as a direct effect of smoke inhalation (157, 158).

In adults, involuntary smoking was linked to a generally increased risk of malignancy and to excess risk at specific sites. Miller (159) interviewed surviving relatives of 537 deceased nonsmoking women in western Pennsylvania concerning the smoking habits of their husbands. A significantly increased risk of cancer death (odds ratio = 1.94, $p < 0.05$) was found in women who were married to smokers and were also not employed outside their homes. The large number of potential subjects who were not interviewed and the possibility of information bias detract from this report.

Sandler and colleagues (160–162) conducted a case-control study on the effects of childhood and adulthood exposures to environmental tobacco smoke on the risk of cancer. The 518 cases included all cancers other than basal cell cancer of the skin; the cases and the matched controls were between 15 and 59 yr of age. For all sites combined, significantly increased risk was found for parental smoking (crude odds ratio = 1.6) and for marriage to a smoking spouse (crude odds ratio = 1.5); the effects of these 2 exposures were independent (162). Significant associations were also found for some individual sites: for childhood exposure (161), maternal and paternal smoking increased the risk of hematopoietic malignancy, and for adulthood exposure (160), spouse's smoking increased the risk for cancers of the female breast, female genital system, and the endocrine system. These findings are primarily hypothesis generating and require replication. In a case-control study, such as reported by Sandler and colleagues, biased information on exposure to environmental tobacco smoke is of particular concern. Hirayama (136) has reported significantly increased mortality from nasal sinus cancers and from brain tumors in nonsmoking women married to smokers in the Japanese cohort. In a case-control study of bladder cancer, involuntary smoke exposure at home and at work did not increase risk (163).

These associations of involuntary smoking with cancer at diverse nonrespiratory sites cannot be readily supported with arguments for biological plausibility. Increased risks at some of the sites, e.g., cancer of the nasal sinus and female breast cancer, have not been observed in active smokers (130). In fact, the International Agency for Research on Cancer has concluded that effects would not be produced in passive smokers that would not be produced to a larger extent in active smokers (152).

Cardiovascular Disease. While extensive data establish active cigarette smoking as a causal risk factor for cardiovascular diseases (164), only a few studies have addressed involuntary smoking as a risk factor for these diseases. In the cohort of nonsmoking Japanese women, Hirayama (165) found a small but statistically significant increased risk of death from ischemic heart disease associated with the husband's smoking. Garland and associates (166) prospectively determined mortality from ischemic heart disease in nonsmoking older women residing in southern California. After adjustment for established risk factors, marriage to a smoking spouse was associated with a relative risk of 2.7 ($p < 0.10$). Gillis and colleagues (120) assessed the baseline prevalence of cardiovascular symptoms and major electrocardiographic abnormalities in a population sample residing in Scotland and then determined cause-specific mortality for up to 10 yr of follow-up. In their preliminary report, involuntary smoking was not associated with the prevalence of cardiovascular symptoms at baseline nor with cardiovascular mortality on follow-up. A case-control study in England did not show increased risk for ischemic heart disease or for stroke in nonsmokers married to smokers (123).

Total Mortality. Two cohort studies, the previously mentioned study in Scotland (120) and an investigation of civil servants and their spouses in Amsterdam (167), provided information on involuntary smoking and mortality from all causes. In the Scottish study, total mortality was increased for women living with a smoker but not for men (120). In contrast, mortality was not increased for nonsmoking female subjects in the study in Amsterdam (167). Neither study directly controlled for other factors that influence total mortality.

Summary. The effects of active smoking and the toxicology of cigarette smoke have been comprehensively examined. When considered in the context of that extensive information, the evidence on involuntary smoking supports conclusions concerning certain health effects. In children, involuntary smoking increases the occurrence of lower respiratory illness during infancy, increases the frequency of chronic respiratory symptoms, and reduces the level of lung function. In adults, involuntary smoking is a cause of lung cancer, but associations with other diseases have not been unequivocally established.

Nitrogen Dioxide

Introduction. Nitrogen dioxide causes lung damage at high concentrations (168, 169), but effects at levels currently encountered in outdoor and indoor air have been difficult to characterize. Early studies focused on the health effects of ambient NO_2 (25). However, in the late 1970s, investigators recognized that indoor NO_2 sources were also contributing to personal exposure and that indoor concentrations often exceeded outdoor concentrations in many homes (15). Consequently, more recent studies have em-

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TABLE 6

EFFECTS OF GAS COOKING ON RESPIRATORY ILLNESSES AND SYMPTOMS IN CHILDREN

Study Population	Outcome Measure	Results
British Studies:		
5,758 children, 6 to 11 yr of age, England and Scotland (178).	Major respiratory symptoms and diseases individually and as a single composite variable describing the presence of any 1 of 6 symptoms or diseases.	Significant associations with gas cooking of selected symptoms and diseases, and of a composite variable.
2,408 children, 42% of original 5,758 in above study (179).	Single composite variable as described above.	Relative risk for composite variable generally exceeded 1.0; risk varied and decreased with age.
4,827 children, 5 to 11 yr of age, England and Scotland (179).	Single composite variable as described above.	Significant effect of gas stoves on composite variable in urban areas only.
808 children, 6 to 7 yr of age, United Kingdom (180).	Single composite variable as described above.	Borderline significant association between composite variable and gas stoves. Increased prevalence as bedroom NO_2 levels increased in a sample with measurements (n = 80).
191 children, 5 to 6 yr of age, England (181).	Single composite variable as described above.	No significant association between bedroom NO_2 levels and prevalence of composite variable.
390 infants, 0 to 1 yr of age, England (182).	Respiratory illnesses and symptoms requiring physician visits, assessed prospectively.	No association between gas stove use and respiratory illnesses and symptoms.
1,565 infants, 0 to 1 yr of age, England (183).	Respiratory illnesses and hospitalizations assessed prospectively to 1 yr.	No significant association between illness or hospitalizations and use of gas for cooking.
Ohio Studies		
441 upper-middle class families including 898 children less than 16 yr of age (184).	Incidence of acute respiratory illness, determined by biweekly telephone calls.	Respiratory illness incidence similar in homes using gas and electric stoves.
180 families from first study, including 178 children less than 12 yr of age (185).	Incidence of acute respiratory illness, determined by biweekly telephone calls and validated by home visits.	Respiratory illness incidence similar in homes using gas and electric stoves.
Harvard Air Pollution Health Study:		
8,120 children, 6 to 10 yr of age, 6 U.S. cities (186, 187).	History of MD diagnosed bronchitis, of serious respiratory illness before age 2, of respiratory illness in last year.	Significant association between current use of gas stove and history of respiratory illness before age 2 (odds ratio = 1.23).
10,106 children, 6 to 10 yr of age, 6 U.S. cities. Expansion of above study (92).	Same as above.	Odds ratio for history of respiratory illness before age 2 decreased to 1.12 (p = 0.07).
Other Studies:		
676 children, 3rd and 4th grades, Arizona (188).	Prevalence of asthma, wheeze, sputum, cough as determined by parent-completed questionnaire.	Significant association between use of gas stove and prevalence of cough (prevalence rate ratio = 1.97).
4,071 children, 5 to 14 yr of age, Pennsylvania (189).	Major respiratory illnesses and symptoms as determined by parent-completed questionnaire.	No significant association between use of gas stove and any symptom or illness variable.
1,138 children, 6 to 12 yr of age, Iowa (91).	Major respiratory symptoms and illnesses as determined by parent-completed questionnaire.	Significant association between current gas stove use and hospitalization for respiratory illness before age 2 (odds ratio = 2.4).
121 children, 0 to 13 yr of age, Connecticut (190).	Number of days of illness.	Number of days of illness associated with average hours of heater use.
231 children, 6 yr of age, Netherlands (191).	Comparison of NO_2 levels in homes of cases (children with asthma) and controls.	NO_2 distributions similar in homes of cases and controls.

phasized sources and effects of indoor NO_2 concentrations.

Exposure. Combustion of gas during cooking and the burning of pilot lights releases nitric oxide (NO), NO_2, CO, CO_2, and water. On average, normal use of an unvented gas cooking range adds 25 parts per billion (ppb) of NO_2 to the background concentration in a home (170). The increase is greater during cold weather when the air exchange rate is usually reduced. During cooking with a gas range, peak levels in the kitchen may reach 200 to 400 ppb (10). Therefore, measured personal exposures to NO_2 are higher for persons living in homes with gas stoves than for persons living in homes with electric stoves (26, 41, 170).

Exposure to NO_2 from gas cooking stoves and ovens is widespread. About 50% of homes in the United States have gas cooking appliances; in some urban areas, such as Los Angeles, more than 90% of homes are equipped with gas appliances (171). The potential importance of NO_2 exposure indoors for health is underscored by comparison of the federal standard set for ambient air, 50 ppb annual average, with levels measured in homes with gas cooking appliances. Sexton and associates (172) used data generated by personal, indoor, and outdoor monitoring to develop a computer model for personal and indoor exposure. The model was applied to residents of 6 U.S. cities. Although none of the cities experienced concentrations above the federal standard in outdoor air, the model predicted that more than 25% of the residents of homes with gas ranges would have annual personal exposures over 50 ppb if ambient NO_2 concentrations averaged 30 ppb.

Health Effects. Most studies of the relationship between residential exposure to NO_2 and health have focused on respiratory symptoms and illnesses and on level of pulmonary function. Experimen-

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TABLE 7

EFFECTS OF GAS COOKING ON LUNG FUNCTION IN CHILDREN

Study Population	Lung Function Measure	Results
808 children, 6 to 7 yr of age, United Kingdom (180).	PEFR, $FEV_{0.75}$, FEF_{25-75}	No association with NO_2 levels or presence of gas stove.
808 children, 0 to 15 yr of age, from 441 families, Ohio (184).	FVC, $FEV_{0.75}$	Data on children not presented separately. No association with presence of a gas stove.
8,120 children, 6 to 10 yr of age, 6 U.S. cities (186, 187).	FVC, FEV_1	Overall reduction of 16 ml and 18 ml, respectively, for FEV_1 and FVC in children from homes with gas stoves.
16,689 children, 6 to 13 yr of age, 7 areas in U.S. (192).	$FEV_{0.75}$	Significant reduction of 19 ml associated with gas stove use in older girls only.
675 children, 3rd and 4th graders, Arizona (188)	FEV_1	No effect of gas stoves on pulmonary level or rate of growth.
[illegible] children, 6 to 12 yr of age, Iowa (91).	FEV_1, FEF_{75}, FEF_{25-75}	No change after isoproterenol challenge in children from homes with gas stoves.
8,720 children, 6 to 10 yr of age, 6 U.S. cities (92).	FEV_1, FVC	Significant reduction in FEV_1, of 0.6% and FVC of 0.7%. Not significant after adjustment for parental education.
[illegible] children, 5 to 14 yr of age, Pennsylvania (193).	FVC, $FEV_{0.75}$, FEF_{25-75}, $Vmax_{75}$, $Vmax_{50}$	No association with use of gas stove.

tal investigations support the choice of these outcome measures; NO_2 may damage the lung directly through its oxidant properties or indirectly by increasing susceptibility to respiratory infections (169, 173). In animal models, NO_2 reduces the efficacy of specific lung defense mechanisms, and effects on mucociliary clearance, the alveolar macrophage, and the immune system have been demonstrated (169, 174, 175).

Data on the health effects of NO_2 concentrations likely to be encountered by the general population are derived from experimental and epidemiologic studies. The results of some human exposure studies imply that levels comparable to those measured in homes may increase airways reactivity in some asthmatics, but the results of other studies are inconsistent (175–177). Although experimental studies are useful for describing effects of controlled exposures, they cannot address the issue of chronic effects from chronic lower level exposures. Numerous epidemiologic investigations have now been carried out to assess their relationship.

The majority of these investigations were cross-sectional surveys of schoolchildren (tables 6 and 7). The investigators generally assessed current symptom status and retrospective illness histories, as obtained by parent-completed questionnaire, and pulmonary function. Although NO_2 levels were measured in several of the investigations (180, 181, 194), exposure was most often assessed by simple questions concerning type of fuel used for cooking. Consistent evidence of excess respiratory symptoms and illnesses in children exposed to gas stoves has not been demonstrated (table 6).

Early reports from two cross-sectional surveys of schoolchildren in Great Britain indicated that children from homes with gas stoves had a higher prevalence of respiratory symptoms than children from homes with electric stoves (178, 179). When one of the survey groups was followed longitudinally, however, the relative risks associated with gas stove use became highly variable and tended to decrease as the children grew older (179). These same British investigators surveyed a third group of 808 schoolchildren, and measured NO_2 concentrations in the homes of a small sample (n = 80 or 103). The prevalence of respiratory symptoms was higher in children from homes where gas was used for cooking and increased with higher bedroom NO_2 concentrations, although both effects were of borderline statistical significance (180). A similar association between measured NO_2 and respiratory symptoms was not replicated, however, when these same investigators subsequently studied another sample of 183 children (181). Two prospective studies of infants in Great Britain also failed to demonstrate an association between the use of gas for cooking and respiratory illness (182, 183).

Data on children from the United States are similarly inconsistent. Two large cross-sectional studies, one involving the Harvard Air Pollution Health Study (186, 187) and the other involving schoolchildren in Iowa (91), have demonstrated that reports of serious respiratory illness before 2 yr of age (186, 187) and hospitalization for respiratory illness before 2 yr of age (91) were more common among children from homes with gas stoves. When the original cohort in the Harvard Air Pollution Health Study was expanded, however, the odds ratio of 1.23 for serious respiratory illness before 2 yr of age decreased to 1.12 (p = 0.07). In the study of Ekwo and associates (91), the effect of exposure to a gas stove varied strongly and inconsistently with parental smoking habits. The effect was absent in homes where 1 parent smoked, largest where both parents smoked, and intermediate where neither smoked. This pattern of interaction cannot be readily interpreted biologically. Schenker and colleagues (189) found no association between type of cooking stove and current respiratory symptoms or previous illness history in a cross-sectional survey of 4,071 schoolchildren in western Pennsylvania.

The relationship between stove type and respiratory illness has also been studied prospectively. Keller and colleagues (184, 185), in a study of 1,952 family members of all ages in Ohio, found that respiratory illness incidence did not vary with stove type. More recently, Berwick and coworkers (190) followed 121 children for 3 months, 59 from homes with kerosene heaters and 62 from homes without such heaters. In a preliminary analysis of their data, they found that hours of heater use, which correlated strongly (r = 0.70) with 1-wk integrated NO_2 measurements, was significantly associated with the occurrence of illness lasting for 1 or more days.

The data concerned with lung function level in children are similarly inconclusive (table 7). Of the 4 investigations with large sample sizes (92, 186, 192, 193), 2 have demonstrated small but statistically significant effects of exposure to a gas stove (186, 192). In initial cross-sectional analysis of data from the Harvard Air Pollution Health Study, Speizer

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TABLE 8

EFFECTS OF GAS COOKING ON PULMONARY ILLNESS, SYMPTOMS, AND FUNCTION OF ADULTS

Study Population	Outcome Measure	Results
441 upper-middle class families, including 1,054 adults over 15 yr, Ohio (184).	Incidence of acute respiratory illness, determined by biweekly telephone calls.	Respiratory illness incidence similar in homes using gas and electric stoves.
120 families from first study, including 289 adults over 18 yr, Ohio (185).	Incidence of acute respiratory illness, determined by biweekly telephone calls and validated by home visit.	Respiratory illness incidence similar in homes with gas and electric stoves.
1,724 adults, ages ≥ 20 yr, Maryland (195).	Major chronic respiratory symptoms, FEV_1, FVC.	Association between gas stove use and increased prevalence of respiratory symptoms, FEV_1 < 80% predicted, FEV_1/FVC < 70%, found in nonsmoking males only.
708 adults, ages ≥ 20 yr. Nonsmoking sample of above population (196).	Major chronic respiratory symptoms, FEV_1, FVC.	Significant association between gas stove use and increased prevalence of chronic cough and phlegm, low FEV_1/FVC.
102 nonsmoking women in lowest quartile of FEV_1 compared to 103 nonsmoking women in highest quartile, Michigan (121).	Comparison of proportions of cases and controls currently using gas stoves.	Marginal association between use of gas stove and lower lung function, (odds ratio = 1.8, p = 0.08).
97 nonsmoking adult females, Netherlands (194).	IVC, FEV, FVC, PEF, MEF_{75}, MEF_{50}, MMEF.	Cross-sectional analysis showed an association between current NO_2 exposure and decreases in most pulmonary function measures. No association with longitudinal decline in pulmonary function.

and associates (186) demonstrated average reductions, adjusted for parental smoking and socioeconomic status, of 16 ml and 18 ml in the FEV_1 and the FVC, respectively, in children from homes with gas stoves compared to children from homes with electric stoves. On expansion of the cohort, however, the reductions in FEV_1 and FVC, although still statistically significant, were 0.6% of predicted for the former and 0.7% for the latter (92). With adjustment for parental education, the effects of exposure to a gas stove were reduced by 30% and were no longer statistically significant. Cross-sectional analysis of lung function data collected at the children's second examination did not show significant effects of stove type. With extension of the follow-up interval, the investigators assessed determinants of pulmonary function growth and found no effect of gas stove exposure (114).

Hasselbad and associates (192) analyzed data from the Environmental Protection Agency's Community Health Environmental Surveillance System. They reported that in girls 9 to 13 yr of age, gas stove exposure decreased $FEV_{0.75}$ by an average of 18 ml after adjustment for parental education level and smoking status. An effect was not observed in girls 6 to 8 yr of age nor in boys 6 to 13 yr of age.

In another large cross-sectional study, Vedal and colleagues (193) examined the effects of stove type on spirometric volumes and flow rates in a sample of 3,175 children ages five to 14 years. With adjustment for parental smoking and socioeconomic status, exposure to a gas stove was not significantly associated with reduced lung function level.

The effects of gas stove exposure on lung function level were assessed in 5 other investigations, but the sample sizes were inadequate for detecting effects of the magnitude found in the larger studies. Keller and colleagues (184) performed spirometry on 1 occasion in a sample of the subjects in their surveillance study. The data were not reported separately for children, and overall there was no effect of stove type. In 1 of the cross-sectional surveys conducted in England, the investigators correlated lung function level with 1-wk measurements of NO_2 in the kitchen and in the childrens' bedrooms (180). With a sample of about 400 children, significant effects of NO_2 were not found. Dodge (188) and Ekwo and associates (91) did not find effects of stove type on lung function measures in their cross-sectional studies. Hosein and Corey (110) examined the influence of 9 indoor factors on $FEV_{1.0}$ in 1,357 nonsmoking white children from 3 U.S. towns. They preliminarily reported that exposure to gas stoves was significantly associated with a 0.148-L reduction in FEV_1 level in boys and 0.75-L in girls.

Only a few investigations provide data on acute and chronic effects of NO_2 exposure indoors on adults (table 8). Prospective studies of acute respiratory illness occurrence have not demonstrated excesses in residents of homes with gas stoves (184, 185, 197). Cigarette smoking and chronic respiratory diseases, potential confounding variables, were not considered in these studies.

Potential chronic effects have also been examined in populations of adults (table 8). Comstock and coworkers (195) reported that gas stove use was associated with a significantly increased prevalence of certain chronic respiratory symptoms and of ventilatory impairment in nonsmoking men, but not in smoking men or in women of either smoking status. A subsequent reanalysis limited to the never and former smokers showed significant increases in chronic cough and phlegm and in the prevalence of low FEV_1/FVC in association with gas stove use in both sexes (196).

In a study of 97 nonsmoking rural women from the Netherlands, personal exposure estimates were created by combining 1-wk measurements of NO_2 with time-activity information (118). The investigators demonstrated a cross-sectional association between lung function level and current NO_2 exposure but failed to show an association between retrospectively estimated exposure to NO_2 and longitudinal decline in pulmonary function during the antecedent 17 yr (194).

Using a case-control design, Jones and associates (121) compared cooking fuel exposures of 20- to 39-yr-old nonsmoking women in the highest and lowest quartiles of the lung function distribution in the Tecumseh Community Health Study. The odds ratio for the effect of cooking with gas on lung function level was 1.82 (p = 0.076).

Lebowitz and colleagues (124, 198, 199) have evaluated acute effects of gas stove exposure on lung function and symptom

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in 229 subjects drawn from 117 Tucson households. The families were sampled from a larger study population to include persons with and without asthma, allergies, and airway obstruction. During a 2-yr period, subjects completed symptom diaries and monitored their peak flow daily. Multivariate analyses indicated adverse effects of gas stoves on symptoms and peak flow rate in asthmatics but not in normal subjects (199). However, the magnitude of the effect is difficult to determine from the available publications.

Recently, Kasuga (200) proposed that the urinary hydroxyproline to creatinine ratio is a valid and sensitive indicator of lung damage from environmental pollutants, including tobacco smoke and NO_2. Hydroxyproline, an amino acid constituent of collagen, is a product of collagen catabolism; therefore, an increase in its excretion reflects an increase in collagen destruction.

Matsuki and associates (115, 201) conducted a cross-sectional study of 820 schoolchildren and their 546 mothers during both a summer and a winter period. They measured subjects' 24-h personal NO_2 exposures with filter badges and collected early morning urine samples for evaluation of the hydroxyproline to creatinine ratio. In multiple regression equations, passive smoking status and personal NO_2 were independent and significant predictors of this ratio in both schoolchildren and adult women in both seasons. Distance from a main road, as a surrogate for exposure to automobile exhaust, was found to be a stronger predictor of the ratio in summer than in winter in schoolchildren and a predictor only during the summer in adult women. A linear relationship was also found between the value of the ratio and the amount of passive exposure to tobacco smoke. Other studies, however, have not shown relationships of the hydroxyproline to creatinine ratio with either passive exposure to tobacco smoke (116) or with active smoking (117). Although the hydroxyproline to creatinine ratio could serve as a useful biochemical indicator of lung injury by NO_2 exposure, further investigations are needed to clarify ambiguities in the available data.

Definitive statements concerning the risk of NO_2 exposure from cooking with gas stoves cannot be made at present. Although many studies have examined respiratory illnesses, respiratory symptoms, and lung function in children and adults, their results are not consistent and are not adequate for establishing a causal relationship. Retrospective illness histories may be inaccurate and their results biased by whether the subjects have symptoms or illness at the time of interview (93). Variations in the characteristics of the study populations and differing endpoints may partly explain the differences among the studies. Confidence limits have not been uniformly presented in the studies on gas stoves, and the results of many of the smaller studies that have been judged as negative are probably consistent with the larger studies that show small effects.

Unfortunately, NO_2 exposures were directly measured in only a few investigations (180, 181, 191, 194), and in all of these the measurements spanned at most 2-wk periods. In the other studies, categorical variables, indicating gas or electric stove use, were employed. However, neither limited area measurements nor variables for stove type tightly predict actual personal exposure (170). Thus, the results of all investigations of the health effects of NO_2 exposure from gas stoves are affected by random misclassification. This type of bias reduces the magnitude of the observed association from the value that would be found if the exposure of subjects was correctly estimated (25). Ozkaynak and associates (202) have shown that misclassification introduced by the use of a categorical variable for stove type may introduce substantial underestimation of the true relative risk values associated with the actual NO_2 exposure.

Bias from inadequate control of confounding factors must also be considered in interpreting the foregoing studies (203). Confounding occurs when the effect of 1 variable on the outcome of interest has not been separated from the effects of other variables. For example, maternal smoking has been associated with reduced lung function level in children. Confounding by maternal smoking could arise in a particular study if mothers of infants living in homes with gas stoves were more likely to smoke. With regard to NO_2 exposure from gas stoves and effects on respiratory illnesses and symptoms, and pulmonary function in children, the potential confounding variables include parental smoking, socioeconomic status, and asthma. Active smoking, occupational exposures, and the presence of chronic respiratory diseases should also be considered in adults. Control of these potentially confounding factors has been variable among published studies (203), and in some studies socioeconomic status has been treated as a confounding factor. However, the effect of socioeconomic status represents a summation of the effects of associated environmental and familial factors, one of which may be gas stove exposure. Thus, control for socioeconomic status may reduce the likelihood of finding an effect of gas stove exposure.

Summary. The findings on NO_2 exposure and respiratory illnesses indicate that the magnitude of the NO_2 effect at concentrations encountered in most U.S. homes is likely to be small. Groups with particularly high exposures, such as the urban poor who heat with ovens and those who heat their homes with kerosene or gas space heaters, have not yet been adequately investigated. The evidence on respiratory symptoms and lung function level in children and adults is also inconclusive. However, because more than half of U.S. homes have gas cooking stoves and childhood respiratory illness is extremely common, even a small effect of gas stoves would assume public health importance. In order to detect associations of the anticipated small magnitude, future investigations should employ direct measurement of exposure, rather than surrogate variables. Infants and other potentially susceptible groups seem the most suitable populations for study. Nevertheless, the epidemiologic evidence implies that clinically relevant effects of NO_2 from gas stoves are uncommon at the concentrations found in most U.S. homes.

Carbon Monoxide

Introduction. Carbon monoxide is a[n] odorless, colorless gas with well-char[acterized] effects on oxygen transpor[t] (204). Carbon monoxide interferes wit[h] oxygen transport by avidly binding to he[moglobin] to form carboxyhemoglobi[n] and by shifting the oxyhemoglobin dis[sociation] curve to the left. It also bind[s] to myoglobin, but the physiologic sig[nificance] of the formation of CO-myo[globin] has not been established (205). Carboxyhemoglobin reduces oxygen de[livery] to tissues, as does the hypoxia o[f] altitude. Tissues with the highest ox[ygen] needs, myocardium, brain, and ex[ercising] muscle, are most affected b[y] the formation of carboxyhemoglobi[n]. Research on the health effects of lowe[r] levels of carbon monoxide exposure ha[s] emphasized consequences for these or[gans], particularly in subjects with dis[eases] that make these organs vulnerabl[e] to reduced oxygen transport.

Exposure. Carbon monoxide has num[erous] sources in the home, the office, an[d] other environments. In the home, emi[ssions] from gas appliances and cigar[ettes]

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smoke, and from vehicles in attached garages may elevate CO levels. During cooking with a gas range, hourly CO concentrations typically range from 2 to 6 ppm and 1-h averages may exceed 12 ppm in conventional homes (28). One-hour CO concentrations in small apartments may reach twice the values in single-family residences. Use of gas stove for heating, a common practice among urban poor in northern climates, may increase CO concentrations to 25 to 50 ppm (206). Cigarette smoking is generally a minor source of CO in homes (64). Other combustion sources in homes are kerosene and gas space heaters (207–209).

Carbon monoxide exposure may also be received in vehicles, particularly when entry routes are available for CO from exhaust (210). During urban commuting, CO levels in cars may average 2 to 5 times the concentrations generally measured in homes and offices and by ambient air monitors (211–213). Offices may be contaminated by vehicle exhaust because of building design problems; high CO levels may result (214).

Health Effects. Most evidence on the health effects of low levels of exposure to carbon monoxide, as generally encountered in indoor environments, has been derived from experimental human exposures. This line of investigation has emphasized disease states that increase susceptibility to reductions of oxygen transport: coronary artery disease, peripheral vascular disease, and chronic obstructive pulmonary disease (204, 215, 216). While the evidence was once considered to indicate adverse effects of CO at low levels in affected persons, much of the data is now controversial.

Although the health effects of low levels of CO exposure are controversial, the problem of CO poisoning by indoor combustion sources has been well described and its dimensions should be recognized by clinicians. The clinical manifestations of CO poisoning primarily reflect the effects of reduced oxygen transport to organs, such as the heart and brain, with high oxygen demand. The neurologic manifestations range from impaired mentation and behavioral alterations to coma (217, 218). Delayed and persistent neurologic sequelae may follow CO poisoning (218). Cardiac effects include arrhythmias and myocardial infarction (217).

The nonspecificity and diversity of the manifestations of CO poisoning have been emphasized (217). In fact, the diagnosis of CO poisoning is frequently delayed while alternative diagnoses are considered. In a series from France, the most common misdiagnoses were food poisoning, psychiatric disorders, cerebrovascular disease, intoxication, and heart disease (219). The finding of retinal hemorrhages on fundoscopic examination should alert the clinician to possible CO poisoning (220, 221). Kelly and Sophocleus (220) reported 12 cases of subacute CO poisoning; retinal hemorrhages were found in each of the 5 patients exposed more than 12 h. The incidence of CO poisoning may rise with increased use of space heaters and woodstoves.

Summary. Carbon monoxide poisoning is a well-documented clinical entity that follows exposure to high levels of CO. Effects of the lower levels of CO exposure generally encountered in indoor environments are controversial at present.

Woodsmoke

Introduction. Since the 1973 oil embargo, there has been a resurgence of residential wood use in the United States. During the decade of the 1970s, the shipment of woodstoves increased 10-fold and the current inventory of woodstoves is estimated to exceed 11 million (222). Residential woodburning typically occurs under oxygen-starved conditions that increase emission rates for CO, respirable particulates, and polycyclic aromatic hydrocarbons. In many communities where woodburning is common, ambient concentrations of these pollutants have increased as a result (223). The use of fireplaces and stoves may potentially result in increased indoor concentrations of smoke components by reentrainment of outdoor air or by direct leakage into indoor air.

Exposure. Few assessments of the impact of woodburning stoves and fireplaces on indoor air quality have been performed. Limited evidence suggests that the rate of pollutant emissions from a wood-burning source depends primarily on the degree of air-tightness of the source. Under proper operating conditions the newer "airtight" residential woodstove is under negative pressure and should not leak combustion by-products into the home. However, under non-airtight operations and during startup, stoking, and reloading, pollutants can be emitted indoors. Traynor and colleagues (224) reported indoor CO concentrations of 0.4 to 2.8 ppm during operation of "airtight" stoves, whereas average levels of 1.8 to 14 ppm occurred during operation of "non-airtight" stoves. For submicron sized particles, indoor concentrations were slightly above background (zero to 30 $\mu g/m^3$) during the use of "airtight" stoves and substantially higher with the "non-airtight" stoves (200 to 1,900 $\mu g/m^3$). Indoor concentrations of 5 polycyclic aromatic hydrocarbons greatly exceeded outdoor levels when the "non-airtight" stove was used (224).

These results are consistent with the findings of a study of personal exposures to respirable particulates in a rural community with substantial woodburning for winter heating (225). Analysis of respirable particulate data collected over 7 days of sampling in 24 homes in Waterbury, Vermont, suggested that homes with airtight woodburning stoves have about 4 $\mu g/m^3$ higher indoor concentrations than do the homes without woodburning stoves (225). The elemental composition of indoor and outdoor particles was examined for 5 of these homes. Using the elements as tracers for wood, automobile exhaust, and other sources of particles, as well as for measuring penetration of ambient air, the investigators confirmed that the increased indoor particle levels were due to woodburning.

Elevated concentrations of pollutants may also be caused by woodburning in fireplaces. Moschandreas and colleagues (63, 226) reported benzo(a)pyrene and respirable particulate levels indoors and outdoors from a series of measurements made in 3 homes, 2 with fireplaces and the third with a woodstove. The outdoor concentrations of benzo(a)pyrene rarely exceeded 1 ng/m^3. The indoor benzo(a)pyrene concentrations were substantially higher than outdoors on days when the woodstove was used, averaging 4.7 ng/m^3 indoors. Benzo(a)pyrene was only measured on 1 woodburning day for 1 home with a fireplace. On this day, the integrated particle samples indoors exceeded 11 ng/m^3 benzo(a)pyrene, while those outdoors were less than 0.5 ng/m^3. Respirable particulates were also elevated in all 3 residences on woodburning days. Levels ranged from 14.3 to 72.5 $\mu g/m^3$ in the home with the woodstove, and were 159.9 and 67.6 $\mu g/m^3$ on 1 woodburning day in each of the homes with a fireplace. The investigators concluded that woodburning in a stove or a fireplace may be an important source of indoor pollution.

In summary, airtight woodstoves contribute relatively low concentrations of particulates, CO, and polyaromatic hydrocarbons to the indoor environment. Woodburning in fireplaces and non-

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airtight woodstoves may lead to substantially higher levels of these pollutants. Nonetheless, woodsmoke is a complex mixture and only a few of its components have been measured in homes. For example, measurements of aldehydes such as acrolein have not been performed during residential woodburning. However, the characteristic smell of woodburning in stoves and fireplaces indicates that odorous compounds, some of which are most likely aldehydes, are released by combustion of wood. Further assessment of the impact of woodburning on indoor air quality is needed to describe fully the range of contaminants produced and their concentrations under differing conditions of operation and combustion.

Health Effects. Limited data have been published concerning the health effects of residential wood combustion. *In vitro* experiments demonstrate that emissions from a woodstove induce sister chromatid exchange (227) and are mutagenic, as assessed by the Ames Salmonella assay (228). Using a rabbit model, Fick and colleagues (229) studied the effects of woodsmoke on pulmonary macrophages. They reported that smoke-exposed rabbits, in comparison with controls, produced significantly more cells of all types on bronchoalveolar lavage, and that the rabbit macrophages exhibited a decrease in adherence, phagocytic rate and bacterial uptake. Macrophage viability and bactericidal processing were not affected. Wong and coworkers (230) evaluated the response of guinea pigs to woodsmoke with repeated CO_2 challenges. After exposure, respiratory frequency decreased and ventilatory response to CO_2 was diminished. These effects were transient and full recovery occurred within 3 days.

Only a few epidemiologic studies on the health effects of woodsmoke have been performed. Studies from less developed countries indicate an association between intense smoke exposure in dwellings and chronic pulmonary disease. In a house-to-house survey of adults more than 20 yr of age in Nepal, Pandey (231) found that chronic bronchitis was equally prevalent in men and women, affecting 18.9%, in contrast to the male preponderance usually observed. Further analysis of the data demonstrated an association between prevalence rates for chronic bronchitis and domestic smoke exposure as measured by the number of hours spent daily near the stove (232). Pandey and colleagues (233) subsequently evaluated respiratory function of 150 women ages 30 to 44 yr from 2 rural villages in Nepal. In cigarette smokers, spirometric test results worsened as reported hours of smoke exposure increased, but a similar effect was not present in nonsmokers.

Master (234) randomly selected 94 New Guinea residents for a health evaluation that included a complete history and physical examination. The prevalence of clinical symptoms or abnormal pulmonary findings was extremely high at all ages; 90% of subjects 40 yr of age and older were affected. Although Master collected only descriptive clinical data and no information on exposures, he attributed the high prevalence of abnormalities to domestic smoke exposure. Based on the findings of a cross-sectional study, Anderson (235) has also suggested that woodsmoke exposure contributes to the development of chronic lung disease in adults in New Guinea.

Respiratory effects of woodsmoke have also been examined in children from less developed countries. Anderson (236) conducted a cross-sectional study and a longitudinal study to assess the effects of woodsmoke pollution on children in New Guinea. He evaluated 1,650 children drawn from 2 contrasting communities, 1 at sea level where wood was not burned and 1 in the highlands where wood was commonly burned. The 2 groups did not differ on spirometric testing, physical examination, or clinical history. He also followed 112 children with differing levels of woodsmoke exposure and did not find a consistent relationship between exposure and respiratory abnormalities during a 30-wk surveillance period. In contrast, Kossove (237) reported that Zulu infants less than 13 months of age with severe lower respiratory tract diseases were twice as likely to have a history of daily heavy smoke exposure as were infants without such disease.

Although these studies implicate domestic smoke exposure as a risk factor for the development of respiratory disease in less developed nations, their results should not be generalized to more developed nations. The exposures are orders of magnitude lower on average in more developed countries than in less developed countries. In the less developed countries, low efficiency stoves are used for long periods of time in small huts with poor ventilation, and agricultural waste and dung are often used as fuel (238). These conditions may lead to particulate and benzo(a)pyrene levels that are 10 to 100 times higher than those found in U.S. homes with woodburning stoves (239).

Data on health effects of residential wood combustion in the United States are sparse. In a case report, Honicky and colleagues (240) described an infant with recurrent hospitalizations for severe lower respiratory tract disease characterized by wheeze and pneumonia. The child improved when hospitalized and then relapsed within 12 h after returning home. After the parents removed their woodstove, the child's illnesses ceased. This case prompted the investigators to conduct a prevalence study of respiratory symptoms in 62 children in Michigan, 31 from homes with and 31 from homes without woodburning stoves (241). Using a standardized questionnaire, interviewers asked parents about their children's respiratory symptoms during the previous winter. Symptoms were classified as present or absent and as mild, moderate, or severe. The proportion of children with moderate or severe symptoms was much greater in the group from homes with woodstoves: 84% of children in this group reported at least 1 severe symptom as compared to 3% of the control group. Parental smoking and socioeconomic status were similar in both groups.

In a study of similar design in Massachusetts, Tuthill (242) retrospectively ascertained episodes of acute respiratory illnesses from January through March from 399 parents of school-age children. In contrast to Honicky's results, use of a woodburning stove was not associated with chronic respiratory disease, symptoms such as fever, sore throat, rhinitis, cough and wheeze, or excess (more than 1) respiratory illness. Differences in study populations, type of wood burned or ascertainment of illness may explain the conflicting results of these studies.

Another potential hazard of woodburning stoves is illustrated by a recent case report of a Wisconsin family that experienced arsenic poisoning (243). Over a 3-yr period, the family displayed a variety of symptoms ranging from rashes and muscle cramps to seizures and loss of consciousness. An environmental evaluation of their house revealed that they were burning plywood treated with a chromium-copper-arsenate mixture in their stove.

Summary. Woodsmoke is a complex mixture of gases and particles that has a wide range of potential respiratory effects. The unconfirmed observations of Honicky and colleagues (241) that woodsmoke causes acute respiratory illnesses and symptoms in U.S. children require further study. Investigations in less developed countries suggest that domestic

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smoke exposure contributes to the development of chronic lung disease. This important hypothesis cannot be tested with sufficient sensitivity in most populations in the United States but should be pursued in appropriate locales. Recurrent severe respiratory disease with no underlying causes in an infant should prompt the clinician to determine whether a woodstove is present in the home. In these situations, a therapeutic trial of discontinuing its use seems warranted.

Addendum

During 1987, several new sources of information on indoor air pollution and health have been published. The 4th International Conference on Indoor Air Quality and Climate was held in August 1987. The proceedings were published by the Institute for Water, Soil and Air Hygiene in Berlin (mailing address: Institut für Wasser-, Boden- und Lufthygiene des Bundesgesundheitsamtes, Corrensplatz 1, D-1000 Berlin 33). The U.S. Environmental Protection Agency report "EPA Indoor Air Quality Implementation Plan" and its appendices provide a comprehensive review. Two new reports on environmental radon are available: "Lung Cancer Risk from Indoor Exposure to Radon Daughters," Publication 50 of The International Commission on Radiological Protection, and the report of the Biological Effects of Ionizing Radiation (BEIR) IV Alpha Committee of the National Academy of Sciences.

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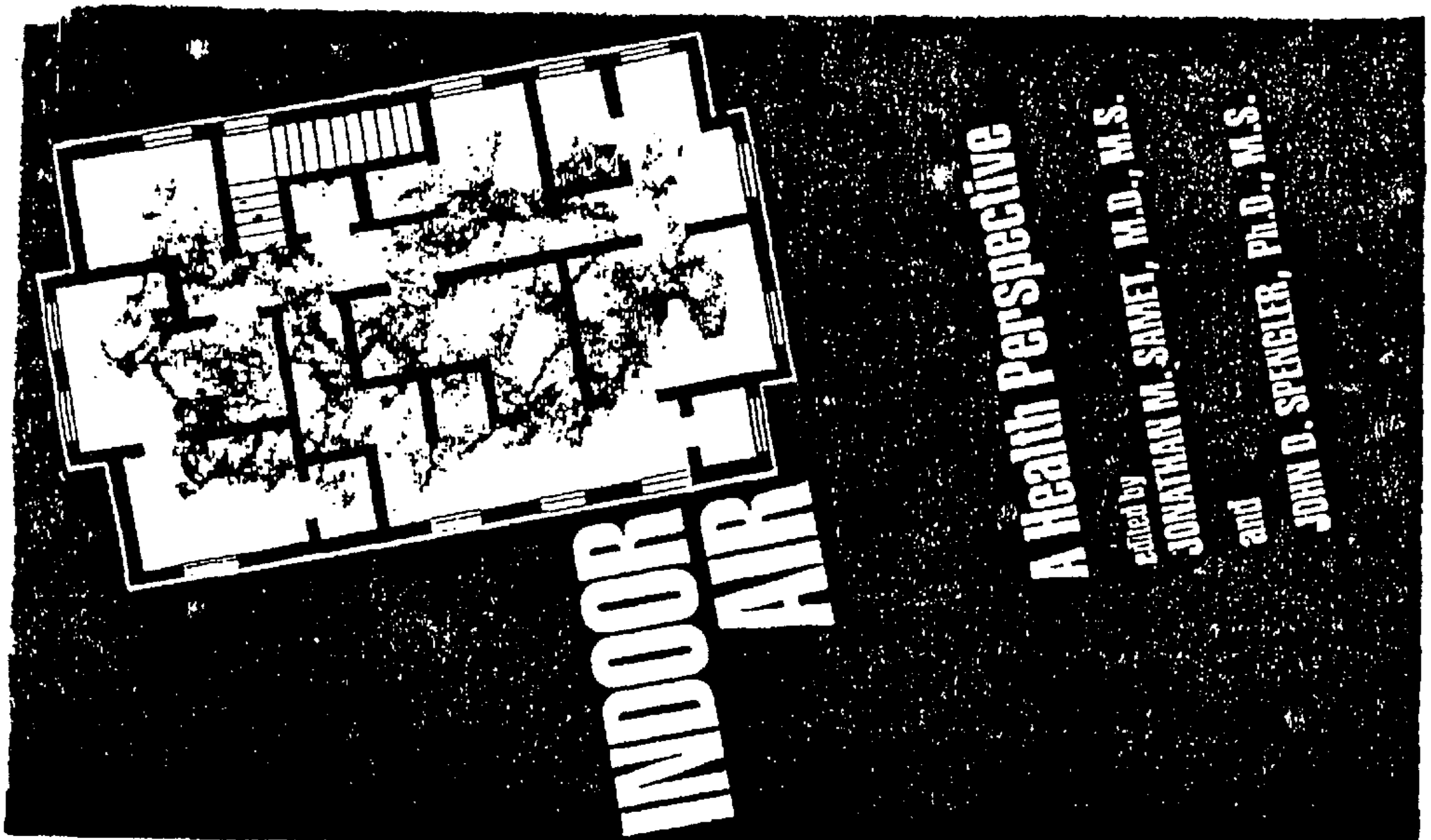

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INDOOR AIR POLLUTION

A Health Perspective

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PREFACE

This book provides a comprehensive review of the rapidly enlarging evidence on indoor air quality and health. The audience for this material continues to grow as the dimensions of the indoor air quality problem become more and more apparent. The issues surrounding indoor air quality are diverse, and the large numbers of specialists involved in the research include epidemiologists, risk assessors, experts in air monitoring, microbiologists, engineers, and psychologists. As the hazards posed by indoor air pollution have been recognized, the evidence on adverse health effects has become increasingly relevant for physicians and other health care providers. The dominant contributions of indoor sources to the total personal exposures for some pollutants have given the subject importance for local, state, and federal regulators. Adverse effects of indoor air pollution have also been the subject of litigation.

The impact of indoor air quality problems on property values and sales has been well demonstrated in the case of radon; it is likely that documentation of adequate indoor air quality for radon and other pollutants may eventually be required at the time of sale of all properties. Such a requirement will bring builders, real estate salespersons, and homeowners into the audience for information on indoor air quality. During the last two decades, outbreaks of illness related to work environments other than industrial settings have been reported with increasing frequency. These outbreaks, often referred to as the "sick-building syndrome" or "tight-building syndrome," have added the building operator and the employer to the list of those who should be concerned about indoor air quality.

A single volume cannot address the needs of so diverse an audience. The intent of this book is to provide a review for the health-oriented readership, and for policymakers, engineers, and lawyers. Unfortunately, the book will not serve the

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relatively small group of researchers who are directly involved with indoor air quality problems.

The structure of this book reflects the complexity of the problem of indoor air quality. The book begins with a perspective on outdoor and indoor air quality. Subsequent chapters address the sources of indoor air pollution, the levels of pollution in homes, the assessment of indoor air quality, and the operating characteristics of buildings. The major topic of the book is the health effects of indoor pollutants. Pollutants that are specifically considered include carbon monoxide, formaldehyde, other volatile organic compounds, environmental tobacco smoke, nitrogen dioxide, radon, and wood smoke. The effects of biologic agents are considered separately and include infectious agents and those agents that cause disease through immunologic mechanisms. Building-related illnesses are also considered in a separate chapter. The topical problem of indoor asbestos is covered in Chapter 2, but the health risks do not receive separate treatment. The occupational hazards of asbestos have been amply described, and direct evidence on the health risks of indoor asbestos is unavailable. The remaining chapters of the book address such other important dimensions of indoor air quality as the control of pollution and its legal aspects. Policy on indoor air is considered in the introductory chapter.

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ACKNOWLEDGMENTS

This book originated with an invitation from Reuben M. Cherniack, editor of the *American Review of Respiratory Disease*, to prepare a "state-of-art" review on indoor air pollution. We accepted and quickly discovered that the task was far larger than we had anticipated. Nevertheless, with our colleague Marian Marbury, we completed the review, which was published in December 1987 and January 1988. Discussions with Zoltan Annau led to an invitation to expand the review into a book for the Johns Hopkins Series in Environmental Toxicology.

During the preparation of this book, we both had support from numerous funding agencies, the availability of which facilitated our work. John D. Spengler derived support from the National Institute of Environmental Health Sciences, the Environmental Protection Agency, the Electric Power Research Institute, the Department of National Health and Welfare Canada, and the Gas Research Institute. Jonathan M. Samet was funded by the Department of Energy, the Health Effects Institute, and the National Heart, Lung, and Blood Institute. Portions of Chapters 2, 6, 7, 9, 10, and 15, in an earlier form, originally appeared in the *American Review of Respiratory Disease* and are reprinted with permission from *ARRD* 1987, Volume 136, and *ARRD* 1988, Volume 137. Portions of Chapter 6, in an earlier form, originally appeared in the *Annals of Sports Medicine* 4 (1988). Portions of Chapter 15, in an earlier form, originally appeared in the *Journal of the National Cancer Institute* 81 (1989). Chapter 17 is being published simultaneously in *Indoor Air Pollution: The Complete Resource Guide*, by the Bureau of National Affairs, Inc.

The contributions of many people were needed to complete this book. The authors worked hard in preparing their chapters and in revising them after review. Rebecca Mosher worked tirelessly in entering the material and preparing illustrations. Dorothy Warner also worked on the volume. Lee Fernando kept everyone organized. Rita Elliott edited the volume. Her diligence in assuring that the volume was readable and that the material was correct deserves special thanks. Wendy Harris remained supportive throughout preparation of the manuscript.

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6

ENVIRONMENTAL TOBACCO SMOKE

Jonathan M. Samet, M.D., M.S., William S. Cain, Ph.D., Brian P. Leaderer, Ph.D., M.P.H.

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Extensive toxicologic, experimental, and epidemiologic data, largely collected since the 1950s, have established that active cigarette smoking is the major preventable cause of morbidity and mortality in the United States (U.S. Department of Health, Education, and Welfare [DHEW] 1979; U.S. Department of Health and Human Services [DHHS] 1989). More recently, involuntary exposure to tobacco smoke has been investigated as a risk factor for disease and also found to be a cause of preventable morbidity and mortality in nonsmokers. The 1986 report of the surgeon general on smoking and health (U.S. DHHS 1986) and a report by the National Research Council (NRC 1986a), also published in 1986, comprehensively reviewed the data on involuntary exposure to tobacco smoke; both reports concluded that involuntary smoking was a cause of disease in nonsmokers.

This chapter will summarize the converging evidence on the health effects of involuntary exposure to tobacco smoke. To date, research on involuntary smoking has focused on respiratory effects although recent investigations have examined associations with diverse conditions including nonrespiratory cancers, ischemic heart disease, age at menopause, sudden infant death syndrome, and birth weight (Table 6.1).

EXPOSURE TO ENVIRONMENTAL TOBACCO SMOKE

CHARACTERISTICS OF ENVIRONMENTAL TOBACCO SMOKE

Nonsmokers inhale environmental tobacco smoke (ETS), the combination of the sidestream smoke that is released from the cigarette's burning end with the mainstream smoke exhaled by the active smoker (First 1985). The inhalation of ETS is generally referred to as *passive smoking* or *involuntary smoking*. The exposures of involuntary and active smoking differ quantitatively and, to some

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Table 6.1 Established and Potential Health Effects of Involuntary Exposure to Tobacco Smoke

Established
Increased lower respiratory infections in children
Increased respiratory symptoms in children
Reduced lung growth in children
Increased lung cancer risk in nonsmokers
Irritation of the eyes, nose, throat, and lower respiratory tract
Potential
Increased respiratory symptoms in adults
Reduced lung function in adults
Increased risk for asthma
Exacerbation of asthma
Increased risk for cardiovascular disease
Increased risk for nonrespiratory cancers
Earlier age at menopause
Increased risk for sudden infant death
Reduced birth weight

extent, qualitatively (NRC 1981, 1986a; Sterling and Kobayashi 1982; U.S. DHHS 1984, 1986). Because of the lower temperature in the burning cone of the smoldering cigarette, most partial pyrolysis products are enriched in sidestream as compared with mainstream smoke. Consequently, sidestream smoke has higher concentrations of some toxic and carcinogenic substances than mainstream smoke does; however, dilution by room air markedly reduces the concentrations inhaled by the involuntary smoker in comparison with those inhaled by the active smoker. Nevertheless, involuntary smoking is accompanied by exposure to many of the toxic agents generated by tobacco combustion (NRC 1981, 1986a; Sterling and Kobayashi 1982; U.S. DHHS 1984, 1986).

ENVIRONMENTAL TOBACCO SMOKE CONCENTRATIONS

Tobacco smoke is a complex mixture of gases and particles which contains myriad chemical species (U.S. DHEW 1979; U.S. DHHS 1984). Not surprisingly, tobacco smoking in indoor environments increases levels of respirable particles, nicotine, polycyclic aromatic hydrocarbons, carbon monoxide (CO), acrolein, nitrogen dioxide (NO_2), and many other substances (Table 6.2). The extent of the increase in concentration varies with the number of smokers, the intensity of smoking, the ventilation rate of the indoor space, and the use of air cleaning devices. Several components of cigarette smoke have been measured in indoor environments as markers of the contribution of tobacco combustion to indoor air pollution. Particles have been measured most often because both sidestream and mainstream smoke contain high concentrations of particles in the respirable size range (NRC 1986a; U.S. DHHS 1986). Particles are a nonspecific marker of tobacco smoke contamination, however, because numerous sources other than

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Table 6.2 Selected Studies of Tobacco Smoke Component Concentrations in Various Environments

Referenced	Location	Component	Mean Concentration
Badre et al. (1978)	Room, [illegible] smokers	Acrolein	0.19 mg/m³
Badre et al. (1978)	Room, [illegible] smokers	Benzene	0.11 mg/m³
Wallace (1987)	New Jersey homes, smokers	Benzene	16 µg/m³, overnight
	New Jersey homes, nonsmokers	Benzene	8.4 µg/m³, overnight
Chappell and Parker (1977)	Office	CO	2.5 ppm, 2- to 3-min samples
Chappell and Parker (1977)	Nightclub	CO	13.0 ppm, 2- to 3-min samples
Hinds and First (1975)	Restaurant	Nicotine	5.2 µg/m³, 2.5-h samples
Hinds and First (1975)	Train	Nicotine	6.3 µg/m³, 2.5-h samples
Muramatsu et al. (1984)	Cafeteria	Nicotine	26.4 µg/m³
Weber and Fischer (1980)	Office	NO_2	24 ppb
Repace and Lowrey (1980)	Cocktail party	Particles	351 µg/m³, 15-min sample
	Bowling alley	Particles	202 µg/m³, 20-min sample
	Bar	Particles	334 µg/m³, 26-min sample
Spengler et al. (1981)	Residence, ≥2 smokers	Particles	70 µg/m³, 24-h sample
	Residence, 1 smoker	Particles	37 µg/m³, 24-h sample
Henderson et al. (1989)	Residence, cigarette smoking	Nicotine	3.4 µg/m³, 14-h sample
	Residence, no cigarette smoking	Nicotine	0.3 µg/m³, 14-h sample

tobacco combustion add particles to indoor air. Studies of levels of ETS components have been conducted largely in public buildings; fewer studies have been conducted in the home and office environments (NRC 1986a; U.S. DHHS 1986).

The contribution of various environments to personal exposure to tobacco smoke varies with the time-activity pattern, the distribution of time spent in different locations. Time-activity patterns may heavily weight exposures in particular environments for certain groups of individuals. For example, exposure in the home predominates for infants who do not attend day-care (Harlos et al. 1987). For adults residing with nonsmokers, the workplace may be the principal location at which exposure takes place.

The contribution of smoking in the home to indoor air pollution has been demonstrated by studies using personal monitoring and monitoring of homes for respirable particles. Spengler et al. (1981) monitored homes in six U.S. cities for respirable particle concentrations over several years and found that a smoker of one pack of cigarettes daily contributed about 20 µg/m³ to twenty-four-hour indoor particle concentrations. In homes with two or more heavy smokers, this study showed that the old twenty-four-hour national ambient air quality standard of 260 µg/m³ for total suspended particulates could be exceeded. Because cigarettes are not smoked uniformly over the day, higher peak concentrations must occur

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when cigarettes are actually smoked. Spengler et al. (1985) measured the personal exposures to respirable particles sustained by nonsmoking adults in two rural Tennessee communities. The mean twenty-four-hour exposures were substantially higher for those exposed to smoke at home, 64 $\mu g/m^3$ for those exposed versus 36 $\mu g/m^3$ for those not exposed.

In several recent studies, small numbers of homes have been monitored for nicotine. In a study of ETS exposure of day-care children, average nicotine concentration during the time that the ETS-exposed children were at home was 3.7 $\mu g/m^3$; in homes without smoking, the average was 0.3 $\mu g/m^3$ (Henderson et al. 1989). Coultas, et al. (1990) measured twenty-four-hour nicotine and respirable particle concentrations in ten homes on alternate days for a week and then on five more days during alternate weeks. The mean levels were comparable to those in the study of Henderson et al. (1989), but some twenty-four-hour values were as high as 20 $\mu g/m^3$. Nicotine and respirable particle concentrations varied widely in the homes.

The total exposure assessment methodology (TEAM) study, conducted by the U.S. Environmental Protection Agency, provided extensive data on concentrations of twenty volatile organic compounds in a sample of homes in several communities (Wallace 1987). Indoor monitoring showed increased concentrations of benzene, xylenes, ethylbenzene, and styrene in homes with smokers compared with homes without smokers.

More extensive information is available on levels of environmental tobacco smoke in public buildings of various types (Table 6.2). Monitoring in locations where smoking may be intense, such as bars and restaurants, has generally shown elevations of particles and other markers of smoke pollution where smoking is taking place (NRC 1986a; U.S. DHHS 1986). For example, Repace and Lowrey (1980) used a piezobalance to sample aerosols in restaurants, bars, and other locations. In the places sampled, respirable particulate levels ranged up to 700 $\mu g/m^3$, and the levels varied with the intensity of smoking. Similar data have been reported for the office environment, although the information is more limited (NRC 1986a; U.S. DHHS 1986).

Transportation environments may also be affected by cigarette smoking. Contamination of air in trains, buses, automobiles, airplanes, and submarines has been documented (NRC 1986a; U.S. DHHS 1986). A recent NRC report (NRC 1986b) on air quality in airliners summarized studies of tobacco smoke pollutants in commercial aircraft. In one study, during a single flight, the NO_2 concentration varied with the number of passengers with a lighted cigarette. In another study, respirable particles in the smoking section were measured at concentrations fivefold or greater than in the nonsmoking section. Peaks as high as 1,000 $\mu g/m^3$ were measured in the smoking section. Mattson and colleagues (1989) used personal exposure monitors to assess nicotine exposures of passengers and flight attendants. All persons were exposed to nicotine, even if seated in the nonsmoking portion of the cabin. Exposures were much greater in the smoking than in the nonsmoking section and were also greater on aircraft with recirculated air.

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BIOLOGICAL MARKERS OF EXPOSURE

Biologic markers of tobacco smoke exposure can be used to describe the prevalence and the dosimetry of involuntary smoking and to validate questionnaire-based measures of exposure. In both active and involuntary smokers, the detection of smoke components or their metabolites in body fluids or alveolar air provides evidence of exposure to tobacco smoke, and levels of these markers can be used to gauge the intensity of exposure. The risks of involuntary smoking have also been estimated by comparing levels of biologic markers in active and involuntary smokers.

At present, the most sensitive and specific markers for tobacco smoke exposure are nicotine and its major metabolite, cotinine (Jarvis and Russell 1984; NRC 1986a; U.S. DHHS 1988). Neither nicotine nor cotinine is present in body fluids in the absence of exposure to tobacco smoke. Because the circulating half-life of nicotine is generally shorter than two hours (Rosenberg et al. 1980), nicotine concentrations in body fluids reflect more recent exposures. In contrast, cotinine has a half-life in the blood or plasma of active smokers which ranges from less than ten hours to about forty hours (Kyerematen et al. 1982; Benowitz et al. 1983; U.S. DHHS 1988); hence, cotinine levels provide information about more chronic exposure to tobacco smoke in both active and involuntary smokers. Whether cotinine has the same half-life in plasma, saliva, and urine is presently controversial, as is the choice of the optimal body fluid for measuring cotinine for research purposes (Jarvis et al. 1988; Wall et al. 1988; Coultas and Samet 1990; Haley, Sepkovic, and Hoffmann 1989). Thiocyanate concentration in body fluids, concentration of CO in expired air, and carboxyhemoglobin level distinguish active smokers from nonsmokers but are not as sensitive and specific as cotinine for assessing involuntary exposure to tobacco smoke (Hoffman et al. 1984a, 1984b; Jarvis and Russell 1984).

Cotinine levels have been measured in adult nonsmokers and in children (Table 6.3). In the studies of adult nonsmokers, exposures at home, in the workplace, and in other settings determined cotinine concentrations in urine and saliva. The cotinine levels in involuntary smokers ranged from less than 1 percent to about 8 percent of cotinine levels measured in active smokers. Smoking by parents was the predominant determinant of the cotinine levels in their children. For example, Greenberg et al. (1984) found significantly higher concentrations of cotinine in the urine and saliva of infants exposed to cigarette smoke in their homes in comparison with unexposed controls. Urinary cotinine levels in the infants increased with the number of cigarettes smoked during the previous twenty-four hours by the mother. In a study of schoolchildren in England, salivary cotinine levels rose with the number of smoking parents in the home (Jarvis et al. 1985).

The results of studies on biologic markers have important implications for research on involuntary smoking and add to the biologic plausibility of associations between involuntary smoking and disease documented in epidemiologic studies. The data on marker levels provide ample evidence that involuntary exposure leads to absorption, circulation, and excretion of tobacco smoke compo-

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Table 6.3 Selected Studies of Cotinine Levels in Nonsmokers

Population	Findings
100 adult patients attending clinics in London (Jarvis et al. 1984)	Cotinine levels parallel self-reported exposure. In nonsmoking, mean = 1.5 ng/ml in saliva; in active smokers, mean = 309.9 ng/ml.
151 adult males attending a clinic in London and 70 subjects from Oxford (Wald et al. 1984, Wald and Ritchie 1984)	Urinary cotinine level increased with reported duration of smoke exposure. In nonexposed nonsmokers, median level = 2.0 ng/ml; exposed nonsmokers, median = 6.0 ng/ml; active cigarette smokers, median = 1,645 ng/ml. Smoking by wife increased cotinine concentrations threefold in nonsmoking men.
51 infants attending clinics in North Carolina (Greenberg et al. 1984)	In nonexposed, median urinary level = 4 ng/mg creatinine; exposed, median = 351 ng/mg creatinine. Salivary creatinine levels higher in exposed.
472 nonsmoking adults in Japan (Matsukura et al. 1984)	Exposure at home and at work independently increased urinary cotinine level. In nonsmokers, median = 680 ng/mg creatinine; active smokers, median = 8,570 ng/mg creatinine.
569 schoolchildren, age 11–16 years, in Bristol, England (Jarvis et al. 1985)	Salivary cotinine level increased with the number of smoking parents. If neither parent smoked, median = 0.20 ng/ml; if both smoked, median = 2.70 ng/ml.
38 children, age 3–15 months, attending a child center in North Carolina (Pattishall et al. 1985)	Serum cotinine level increased with the number of smokers in the home. In children without household exposure, mean = 1.0 ng/ml; exposed, mean = 4.1 ng/ml.
839 children and adults in a population sample in New Mexico (Coultas et al. 1987)	Salivary cotinine level increased with the number of smokers in the home. If no smokers in the home, median level was zero. If one or more smokers, median was greater than zero and increased with the number of smokers
260 adult municipal workers in Dallas, Texas (Haley et al. 1989)	Urinary cotinine level about 2.5 times greater for persons living with smokers. Somewhat greater levels for those exposed at work.

nents. The studies of biologic markers also confirm the high prevalence of involuntary smoking, as ascertained by questionnaire (Coultas et al. 1987). The observed correlations between reported exposures and levels of markers suggest that questionnaire methods for assessing recent exposure have some validity.

Comparisons of levels of biologic markers in smokers and nonsmokers have been made in order to estimate the relative intensities of active and involuntary smoking. However, proportionality cannot be assumed between the ratio of the levels of markers in passive and active smokers and the relative doses of other tobacco smoke components. Nonetheless, several investigators have attempted to characterize involuntary smoking in terms of active smoking. For example, Foliart and colleagues (Foliart, Benowitz, and Becker 1983) measured urinary excretion of nicotine in flight attendants during an eight-hour flight and estimated that the

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average exposure was 0.12–0.25 mg of nicotine. Russell. West, and Jarvis (1985) compared nicotine levels in nonsmokers exposed to tobacco smoke with levels achieved following infusion of known doses of nicotine. On the basis of this comparison the investigators estimated that the average rate of nicotine absorption was 0.23 mg/h in a smoky tavern, 0.36 mg/h in an unventilated smoke-filled room, and 0.014 mg/h from average daily exposure. In active smokers the first cigarette of the day resulted in absorption of 1.4 mg of nicotine.

HEALTH EFFECTS OF INVOLUNTARY SMOKING

LOWER RESPIRATORY TRACT ILLNESSES IN CHILDHOOD

Epidemiologic investigations have linked involuntary smoking in children to increased occurrence of lower respiratory tract illness during infancy and childhood; presumably these illnesses are infectious in etiology and do not represent a direct response of the lung to toxic components of ETS. Investigations conducted throughout the world have demonstrated an increased risk of lower respiratory tract illness in infants with smoking parents (Table 6.4). These studies indicate a significantly increased frequency of bronchitis and pneumonia during the first year of life of children with smoking parents. Although the health outcome measures varied somewhat among the studies, the relative risks associated with involuntary smoking were similar, and dose-response relationships with the extent of parental smoking were demonstrable. Although most of the studies have shown that maternal smoking rather than paternal smoking underlies the increased risk, a recent study from China shows that paternal smoking alone can increase the incidence of lower respiratory illness (Yue Chen, Wanxian, and Shunzhang 1986). In these studies (Table 6.4), an effect of passive smoking has not been readily identified after the first year of life. The strength of the effect of passive smoking during the first year of life may reflect higher exposures consequent to the time-activity patterns of young infants, which place them in close proximity to cigarettes smoked by their mothers.

For school-age children, parental smoking also increases the occurrence of chest illnesses. For example, in a survey of 4,071 Pennsylvania children ages five to fourteen years, the proportion of children having a chest illness of three days or more during the past year rose from 8.8 to 11.8 to 13.6 percent with none, one, and two smoking parents, respectively (Schenker, Samet, and Speizer 1983). The findings in other large surveys have been similar (Ware et al. 1984; Somerville, Rona, and Chinn 1988).

RESPIRATORY SYMPTOMS AND ILLNESSES IN CHILDREN

Data from numerous surveys demonstrate a greater frequency of the most common respiratory symptoms, cough, phlegm, and wheeze in the children of smokers (U.S. DHHS 1986). In these studies the subjects have generally been schoolchildren, and the effects of parental smoking have been examined. Thus, the less

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Table 6.4 Early Childhood Respiratory Illnesses and Involuntary Smoking

Study Population	Study Design	Effect of Involuntary Smoking	Comments
10,672 births in Israel, 1967–68 (Harlap and Davies 1974)	Antenatal maternal smoking history, monitoring of admissions during first year of life	Significant increase in hospitalization for pneumonia and bronchitis; RR* = 1.4	Dose-response relationship present, maternal smoking only
2,205 births in England, 1963–65 (Leeder et al. 1976)	Prospective cohort with annual questionnaire	Significant increase in bronchitis or pneumonia in first year of life; RR = 1.7 if one parent smoked, RR = 2.6 if both smoked	Sex of smoking parent not examined
12,068 births in Finland, 1966 (Rantakallio 1978)	Prospective cohort with follow-up of hospitalizations, physician visits, and mortality	Significant increase of hospitalization for respiratory diseases during first 5 years, RR = 1.7	Effect largest during first year; maternal smoking only and measured during pregnancy
1,265 births in New Zealand, 1977 (Fergusson and Horwood 1985)	Prospective cohort with diaries, physician, and hospital record review	Significant increase in bronchitis or pneumonia during the first year of life; RR = 2.0, if mother smoked; dose-response present in first	No effect of paternal smoking; effect of maternal smoking equivocal in second year, absent in third
1,565 infants in Scotland, 1980 (Ogston, Florey, and Walker 1985, 1987)	Prospective cohort with record review and interview at one year of age	Significant increase in report of a respiratory illness during the first year of life; RR = 1.54 if either parent smoked	Incidence lowest for group with mother alone smoking
130 children with RS virus infection in infancy, England (Pullan and Hey 1982)	Case-control with 111 controls, performed 10 years after index illness	Significant effect of maternal smoking at time of illness; RR = 3.2	Effect of paternal smoking also present
29 children hospitalized with respiratory syncytial virus infection before age 2 years, and 58 controls, New York (Hall et al. 1984)	Case-control study with controls hospitalized for nonrespiratory condition	Significant effect of smoking in the household; odds ratio = 4.8	Smoking by specific parents not considered

*RR = relative risk. Calculated from published data if not provided by the authors.

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prominent effects of passive smoking, in comparison with the studies of lower respiratory illness in infants, may reflect lower exposures to ETS by older children who spend less time with their parents.

Recent results from several large studies provide convincing evidence that involuntary exposure to tobacco smoke increases the occurrence of cough and phlegm in the children of smokers although earlier data from smaller studies had been ambiguous (Table 6.5). In a study of ten thousand schoolchildren in six U.S. communities, smoking by parents increased the frequency of persistent cough in their children by about 30 percent (Ware et al. 1984). The effect of parental smoking derived primarily from smoking by the mother. Charlton (1984) conducted a survey on cigarette smoking which included 15,709 English children, ages eight to nineteen years. In the nonsmoking children, the prevalence of frequent cough was significantly higher if either the father or the mother smoked. In another large study in the United Kingdom, parental smoking was associated with day or night cough in children aged five to eleven years (Somerville, Rona, and Chinn 1988).

For the symptom of chronic wheeze, the preponderance of the evidence also indicates an excess associated with involuntary smoking (Table 6.5). In a survey of 650 schoolchildren in Boston, persistent wheezing was the most frequent symptom (Weiss et al. 1980); the prevalence of persistent wheezing increased significantly as the number of smoking parents increased. In the large study of children in six U.S. communities, the prevalence of persistent wheezing during the previous year was significantly increased if the mother smoked (Ware et al. 1984). In English children aged five to eleven years, parental smoking was associated with a

Table 6.5 Prevalence of Respiratory Symptoms in Selected Investigations of Children, by Number of Smoking Parents

Subjects	Respiratory Symptoms	Prevalence (per 100) by Number of Smoking Parents 0	1	2
2,426 children, aged 6–14, England (Colley 1974)	Chronic cough	15.6	17.2	22.2
3,105 nonsmoking children, aged 12–13, England (Bland et al. 1978)	Cough during the day or at night	16.4	19.0	23.5
650 children, aged 5–9, Massachusetts (Weiss et al. 1980)	Chronic cough and phlegm	1.7	2.7	3.4
	Persistent wheeze	1.8	6.8	11.8
4,071 children, aged 5–14, Pennsylvania (Schenker, Samet, and Speizer 1983)	Chronic cough	6.3	7.0	8.3
	Chronic phlegm	4.1	4.8	4.0
	Persistent wheeze	7.2	7.7	5.4
8,528 children, aged 5–9, six U.S. cities (Ware et al. 1984)	Chronic cough	7.7	8.4	10.6
	Persistent wheeze	9.9	11.0	13.1
1,733 nonsmoking children, aged 8–10, England (Charlton 1984)	Frequent cough (boys)	35	42	48
	Frequent cough (girls)	32	40	52

Source: abstracted from U.S. DHHS (1986, Table 4, pp. 360–62) and from Charlton (1984)

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more frequent report of persistent wheezing (Somerville, Rona, and Chinn 1988). The odds ratio for exposure to twenty cigarettes per day at home was 1.6 (95 percent confidence interval 1.2–2.2) compared with no exposure. McConnochie and Roghmann (1986) assessed predictors of wheeze in a retrospective study of children who had mild bronchiolitis in infancy and of control children. At a mean age of 8.3 years, current exposure to tobacco smoke increased the risk of wheezing (relative risk = 1.9). Parental smoking predicted the occurrence of wheezing at age five years in a study of children at risk for atopy (Cogswell, Mitchell and Alexander 1987). Passive smoking has not been significantly associated with wheezing in all studies, however (Leeder et al. 1976; Schenker, Samet, and Speizer 1983; Tashkin et al. 1984).

Although involuntary exposure to tobacco smoke is associated with the symptom of wheeze, evidence for association of involuntary smoking with childhood asthma is conflicting. Exposure to ETS might cause asthma as a long-term consequence of the increased occurrence of lower respiratory infection in early childhood or through other mechanisms (Samet, Tager, and Speizer 1983; Tager 1988). Gortmacker et al. (1982) collected data from two U.S. population samples. The relative risk for asthma associated with maternal smoking was 1.5 in a midwestern urbanized county and 1.8 in a more rural eastern county. In a longitudinal study of children in Tecumseh, Michigan, parental smoking increased the prevalence of asthma at the first examination and was associated with a doubling of the risk for developing asthma during a fifteen-year follow-up period (Burchfiel 1984). A case-control study in the United Kingdom also showed increased risk for asthma in association with exposure to ETS at home (Kershaw 1987). In contrast, in a longitudinal study in New Zealand, parental smoking habits were not found to affect the incidence of asthma during the first six years of life (Horwood, Fergusson, and Shannon 1985). In another longitudinal study of more than two thousand children in Harrow, England, parental smoking was not a significant predictor of the development of asthma during a five-year follow-up period (Leeder et al. 1976). Some cross-sectional studies have also not demonstrated a relationship between parental smoking and asthma (Schenker, Samet, and Speizer 1983; Tashkin et al. 1984; Somerville, Rona, and Chinn 1988). The inconsistencies among these investigations cannot be readily explained.

Although involuntary exposure to tobacco smoke has not been established as a cause of asthma, recent evidence indicates that involuntary smoking worsens the status of those with asthma. Murray and Morrison (1986) evaluated ninety-four asthmatic children of ages seven to seventeen years. Level of lung function, symptom frequency, and responsiveness to inhaled histamine were adversely affected by maternal smoking. In a study of twenty-one asthmatics, ages six to twenty-one years, O'Connor and colleagues (1987) also found that maternal smoking increased the level of airways responsiveness. Male nine-year-old Italian schoolchildren had greater bronchial responsiveness if their parents smoked; the association with smoking was strongest for children with asthma (Martinez et al. 1988). The increased level of airways responsiveness associated with maternal

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smoking would be expected to increase the clinical severity of asthma. In this regard, exposure to smoking in the home has been shown to increase the number of emergency room visits made by asthmatic children (Evans et al. 1987).

Recent studies have also shown that children exposed to cigarette smoke in their homes are at increased risk for middle ear disease. Both acute otitis media (Pukander et al. 1985) and persistent middle ear effusions (Kraemer et al. 1983; Black 1985; Iversen et al. 1985; Strachan, Jarvis, and Feyerabend 1989) have been associated with involuntary smoking.

LUNG FUNCTION IN CHILDREN

On the basis of the primarily cross-sectional data available at the time, the 1984 report of the surgeon general (U.S. DHHS 1984) concluded that the children of smoking parents in comparison with those of nonsmokers had small reductions of lung function, but the long-term consequences of these changes were regarded as unknown. In the two years between the 1984 and the 1986 reports, sufficient longitudinal evidence accumulated to support the conclusion in the 1986 report (U.S DHHS 1986) that involuntary smoking reduces the rate of lung function growth during childhood. New cross-sectional studies have continued to confirm the evidence reviewed in the 1984 report of the surgeon general (Burchfiel 1984; Tashkin et al. 1984; Tsimoyianis et al. 1987), although not all studies have shown adverse effects of involuntary smoking on the lung function of children (Hosein and Corey 1984; Lebowitz, Knudson, and Burrows 1984).

The effects of involuntary smoking on lung growth have been demonstrated in three separate longitudinal studies. Based on cross-sectional data from children in East Boston, Massachusetts, Tager and co-workers (1979) reported in 1979 that the level of FEF_{25-75}, a spirometric flow rate sensitive to subtle effects on airways and parenchymal function, declined with increasing number of smoking parents in the household. In 1983, this investigative group reported the results obtained on follow-up of these children over a seven-year period (Tager et al. 1983). Using a multivariate technique, the investigators showed that both maternal smoking and active smoking by the child reduced the growth rate of the one-second forced expiratory volume (FEV_1). Lifelong exposure of a child to a smoking mother was estimated to reduce growth of the FEV_1 by 10.7, 9.5, and 7.0 percent after one, two, and five years of follow-up, respectively.

Recent longitudinal data from the study in six U.S. cities also showed reduced growth of the FEV_1 in children whose mothers smoked cigarettes (Berkey et al. 1986). The growth rate of the FEV_1 from ages six through ten years was calculated for 7,834 white children. From ages six through ten years, the findings of a statistical analysis were that FEV_1 growth rate is reduced by 0.17 percent per pack of cigarettes smoked daily by the mother. This effect was somewhat smaller than that reported by Tager et al. (1983), although if extrapolated to age twenty years, a cumulative effect of 2.8 percent is predicted.

Burchfiel and co-workers (Burchfiel 1984; Burchfiel et al. 1986) examined the effects of parental smoking on fifteen-year lung function change in subjects in

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the Tecumseh study, who had been enrolled at ages ten through nineteen years. In the female subjects who remained nonsmokers across the follow-up period, parental smoking did not affect lung function change. In nonsmoking males, parental smoking reduced the growth of the FEV_1, forced vital capacity (FVC), and maximum flow rate at 50 percent of FVC (V_{max50}) although the sample size was limited, and the effects were not statistically significant. For the FEV_1 in males, the analysis estimated 7.4 percent and 9.4 percent reductions in fifteen-year growth associated with one or two smoking parents, respectively.

RESPIRATORY SYMPTOMS AND ILLNESSES IN ADULTS

Only a few cross-sectional investigations provide information on the association between respiratory symptoms in nonsmokers and involuntary exposure to tobacco smoke. These studies have only considered exposure to a smoking spouse and have not evaluated sources of exposure outside the home. Consistent evidence of an effect of passive smoking on respiratory symptoms in adults has not been found (Lebowitz and Burrows 1976; Schilling et al. 1977; Comstock et al. 1981; Schenker, Samet and Speizer 1982; Kauffmann et al. 1989).

Neither epidemiologic nor experimental studies have established the role of ETS in exacerbating asthma in adults. The acute responses of asthmatics to ETS have been assessed by exposing persons with asthma to tobacco smoke in a chamber. This experimental approach cannot be readily controlled because of the impossibility of blinding subjects as to exposure to ETS. However, suggestibility does not appear to underlie physiologic responses of asthmatics to ETS (Urch et al. 1988). Of three studies involving exposure of unselected asthmatics to ETS, only one showed a definite adverse effect (Shephard et al. 1979; Dahms, Bolin, and Slavin 1981; Wiedemann et al. 1986). Stankus et al. (1988) recruited twenty-one asthmatics who reported exacerbation with exposure to ETS. With challenge in an exposure chamber, seven of the subjects experienced a more than 20 percent decline in FEV_1.

LUNG FUNCTION IN ADULTS

With regard to involuntary smoking and lung function in adults, exposure to passive smoking has been associated with reduction of the FEF_{25-75} in cross-sectional investigations. White and Froeb (1980) compared spirometric test results in middle-aged nonsmokers with at least twenty years of involuntary smoking in the workplace with the results in an unexposed control group of nonsmokers. The mean FEF_{25-75} of the exposed group was significantly reduced, by 15 percent of predicted value in women and by 13 percent in men. This investigation has been intensely criticized with regard to the spirometric test procedures, the determination and classification of exposures, and the handling of former smokers in the analyses.

A subsequently reported investigation in France examined the effect of marriage to a smoker in more than 7,800 adults in seven cities (Kauffmann, Tessier, and Oriol 1983). The study included 849 male and 826 female nonsmokers exposed to

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tobacco smoking by their spouses' smoking. At more than age forty years, the [illegible] was reduced in nonsmoking men and women with a spouse who smoked. The investigators interpreted this finding as representing a cumulative adverse effect of marriage to a smoker. In a subsequent report, the original findings in the French women were confirmed, but a parallel analysis in a large population of U.S. women did not show effects of involuntary smoking on lung function (Kauffmann et al. 1989).

The results of an investigation of 163 nonsmoking women in The Netherlands also suggested adverse effects of tobacco-smoke exposure in the home on lung function (Brunekreef et al. 1985; Remijn et al. 1985). Cross-sectional analysis of spirometric data collected in 1982 demonstrated adverse effects of tobacco smoke exposure in the home, but in a sample of the women, domestic exposure to tobacco smoke was not associated with longitudinal decline of lung function during the period 1965 to 1982.

Svendsen and co-workers (1987) assessed the effects of spouse smoking on 1,400 nonsmoking male participants in the Multiple Risk Factor Intervention Trial (MRFIT). The subjects were aged thirty-five to fifty-seven years at enrollment and were at high risk for mortality from coronary artery disease. At the baseline visit, the maximum FEV_1 was approximately 100 milliliters lower for the men married to a smoker.

Masi and colleagues (1988) evaluated the lung function of 293 young adults using spirometry and measurement of the diffusing capacity and lung volumes. The results varied with gender. In men, reduction of the maximal mid-expiratory flow rate was associated with maternal smoking and exposure to ETS during childhood. In women, reduction of the diffusing capacity was associated with exposure to ETS at work.

Other studies have not shown chronic effects of involuntary exposure to tobacco smoke in adult nonsmokers. In two cross-sectional studies, marriage to a smoker was not significantly associated with reduction of ventilatory function (Schilling et al. 1977; Comstock et al. 1981). Jones and co-workers (Jones et al. 1983) conducted a case-control study of twenty- to thirty-nine-year-old nonsmoking women in the longitudinal study in Tecumseh. Subjects from the highest and lowest quartiles of the lung function distribution had comparable exposure to smokers in the home. In a study conducted in Germany, the effects of involuntary and active smoking were examined in a population of 1,351 white-collar workers (Kentner, Triebig, and Weltle 1984). Self-reported exposure to ETS at home and at work was not associated with reduction of spirometric measures of lung function.

A conclusion cannot yet be reached on the effects of ETS exposure on lung function in adults. However, further research is warranted because of widespread exposure in workplaces and homes.

LUNG CANCER

In 1981, reports were published from Japan (Hirayama 1981) and from Greece (Trichopoulos et al. 1981) that indicated increased lung cancer risk in nonsmoking

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women married to cigarette smokers. Subsequently, this controversial association has been examined in investigations conducted in the United States, Scotland, Japan, Hong Kong, Sweden, and China. The association of involuntary smoking with lung cancer derives biologic plausibility from the presence of carcinogens in sidestream smoke and the lack of a documented threshold dose for respiratory carcinogenesis in active smokers (U.S. DHHS 1982).

Time trends of lung cancer mortality in nonsmokers have been examined with the rationale that temporally increasing exposure to ETS should be paralleled by increasing mortality rates (Enstrom 1979; Garfinkel 1981). These data provide only indirect evidence on the lung cancer risk associated with involuntary exposure to tobacco smoke. Epidemiologists have tested directly the association between lung cancer and involuntary smoking utilizing conventional designs: the case-control and cohort studies. In a case-control study, the exposures of nonsmoking persons with lung cancer to ETS are compared with those of an appropriate control group. In a cohort study, the occurrence of lung cancer over time in nonsmokers is assessed in relationship to involuntary tobacco smoke exposure. The results of both study designs may be affected by inaccurate assessment of exposure to ETS, by inaccurate information on personal smoking habits that leads to classification of smokers as nonsmokers, and by the misdiagnosis of a cancer of another site as a primary cancer of the lung.

Methodologic investigations suggest that accurate information can be obtained by interview in an epidemiologic study on the smoking habits of a spouse (i.e., never or ever smoker) (Pron et al. 1988; Coultas, Peake, and Samet 1989; Cummings et al. 1989). However, information concerning quantitative aspects of the spouse's smoking is reported with less accuracy. Misclassification of current or former smokers as never smokers may introduce a positive bias because of the concordance of spouse smoking habits (Lee 1988). The extent to which this bias explains the numerous reports of association between spouse smoking and lung cancer is controversial (Wald et al. 1986; Lee 1988).

The evidence from the case-control and the cohort studies does not uniformly indicate increased lung cancer risk in persons exposed to ETS, but most of the studies indicate increased risk in nonsmokers married to smokers (Table 6.6). Hirayama's (1981) early report was based on a prospective cohort study of 91,540 nonsmoking women in Japan. Standardized mortality ratios for lung cancer increased significantly with the amount smoked by the husbands. The findings could not be explained by confounding factors and were unchanged when follow-up of the study group was extended (Hirayama 1984). Based on the same cohort, Hirayama has also reported significantly elevated standardized mortality ratios for lung cancer of 2.1 and 2.3 in nonsmoking men with wives smoking one to nineteen cigarettes and twenty or more cigarettes daily, respectively (Hirayama 1984).

In 1981, Trichopoulos et al. (1981) also reported increased lung cancer risk in nonsmoking women married to cigarette smokers. These investigators conducted a case-control study in Athens, Greece, which included cases with a diagnosis other than adenocarcinoma or bronchioloalveolar carcinoma and controls selected

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Table 6.6 Cohort and Case-Control Studies of Passive Exposure to Tobacco Smoke and Lung Cancer

Study	Findings	Comments
Prospective cohort study in Japan of 91,540 nonsmoking females, 1966–81 (Hirayama 1981)	Age-occupation-adjusted SMRs,[a] by husband smoking: Nonsmokers = 1.00 Ex-smokers = 1.36 Current smokers <20/day = 1.45 >20/day = 1.91	Trend statistically significant; all histologies
Prospective cohort study in the U.S. of 176,139 nonsmoking females, 1960–72 (Garfinkel 1981)	Age-adjusted SMRs, by husband smoking: Nonsmokers = 1.00 Current smokers <20/day = 1.27 >20/day = 1.10	All histologies; effect of husband smoking not significant
Prospective cohort study in Scotland of 8,128 males and females, 1972–82 (Gillis et al. 1984)	Age-adjusted mortality ratios for domestic exposure: Males = 3.25 Females = 1.00	Preliminary, small numbers of cases
Case-control study in Greece of 40 nonsmoking female cases, 149 controls, 1978–80 (Trichopoulos et al. 1981; Trichopoulos, Kalandidi, and Sparros 1983)	Odds ratios by husband smoking: Nonsmokers = 1.0 Exsmokers = 1.8 Current smokers <20/day = 2.4 >20/day = 3.4	Trend statistically significant; histologies other than adenocarcinoma and bronchioloalveolar carcinoma
Case-control study in Hong Kong of 84 female cases and 139 controls, 1976–77 (Chan et al. 1979; Chan and Fung 1982)	Crude odds ratio of 0.75 associated with smoking spouse	All histologies; two reports are inconsistent on the exposure variable
Case-control study in the U.S. with 22 female and 8 male nonsmoking cases, 133 female and 180 male controls (Correa et al. 1983)	Odds ratios by spouse smoking: Nonsmokers = 1.00 <40 pack years = 1.48 ≥41 pack years = 3.11	Significant increase for >41 pack years; bronchioloalveolar carcinoma excluded
Case-control study in the U.S. 25 male and 53 female nonsmoking cases with matched controls, 1971–80 (Kabat and Wynder 1984)	Odds ratio not significantly increased for current exposure at home: Males = 1.26 Females = 0.92	All histologies; findings negative for spouse smoking variable as well
Case-control study in Hong Kong with 88 nonsmoking female cases, 1981–82 (Koo, Ho, and Saw 1984; Koo, Ho, and Lee 1985)	Odds ratio of 1.24 ($p > .40$) for combined home and workplace exposure; no association with cumulative hours of exposure	All histologies
Case-control study in the U.S. with 31 nonsmoking and 189 smoking female cases (Wu et al. 1985)	No significant effects of exposure from parents, spouse, or workplace in smokers and nonsmokers	Adenocarcinoma and squamous cell carcinoma only
Case-control study in the U.S. with 134 nonsmoking female cases (Wu et al. 1985)	Nonsignificant odds ratio of 1.22 if husband smoked; significantly increased odds ratio of 2.11 if husband smoked 20	All histologies; careful exclusion of smokers from the case group

(continued)

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Table 6.6 (*Continued*)

Study	Findings	Comments
	or more cigarettes daily at home; significant trend with number of cigarettes smoked *at home by the husband*	
Case-control study in England with 15 male and 32 female nonsmoking cases, and 30 male and 66 female nonsmoking controls (Lee, Chamberlain, and Alderson 1986)	Overall odds ratio for spouse smoking of 1.1	Hospital-based study
Case-control study in Japan with 19 male and 94 female nonsmoking cases, and 110 male and 270 female nonsmoking controls (Akiba, Kato, and Blot 1986)	For females, odds ratio of 1.5 if husband smoked, for males, odds ratio of 1.8 if wife smoked	Clinical or radiologic diagnosis for 43%, all histologies
Case-control study in Louisiana, Texas, and New Jersey with 99 nonsmoking cases and 736 controls (Dalager et al. 1986)	Adjusted odds ratio for marriage to a smoking spouse was 1.5	Nearly 100% histologic confirmation, all histologies
Case-control study in New Mexico with 28 nonsmoking cases and 292 nonsmoking controls (Humble, Samet, and Pathak 1987)	Adjusted odds ratio for marriage to a smoking spouse was 3.2; no effect in active smokers	All histologies other than bronchiolo-alveolar carcinoma
Case-control study in Sweden with 77 nonsmoking cases, two matched control series (Pershagen, Hrubec, and Svensson 1987)	Odds ratio for marriage to a smoker was 3.3 for squamous and small cell carcinomas	No effect of exposure for other types; study based within a cohort
Case-control study in Colorado with 102 adenocarcinoma cases, 50 males and 52 females, and 131 controls (Brownson et al. 1987)	No effect in entire group; in nonsmoking women, odds ratio of 1.7 for exposure ≥4 h/day	Passive smoking effect not significant in nonsmoking women but only 19 such cases included
Case-control study in Hong Kong with 199 never smoking female cases and 335 controls (Lam et al. 1987)	Overall odds ratio = 1.7, significantly increased for marriage to a smoker; odds ratio = 2.1 for adenocarcinoma	All histologies; no evidence for exposure response
Case-control study in Shanghai with 246 nonsmoking female cases and 375 controls (Gao et al. 1987)	Overall odds ratio = 0.9 for ever living with a smoker; risk increased with duration of living with a smoking husband	All histologies, but majority adenocarcinoma; no effect of childhood exposure
Case-control study in Japan with 90 nonsmoking female cases and 163 controls (Shimizu et al. 1988)	Odds ratio for husband's smoking was 1.1; no effect of exposure at work	All histologies; increased risk from other household members' smoking
Case-control study in Japan with 28 smoking female cases and	Overall odds ratio = 2.3 for marriage to a smoker	Risk increased with the number of

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Table 6.6 (*Continued*)

Study	Findings	Comments
62 controls (Inoue and Hirayama 1988)		cigarettes smoked by the husband
Case-control study in Tianjin China with 54 nonsmoking female cases and 93 controls (Geng et al. 1987)	Overall odds ratio = 2.2 for marriage to a smoker	All histologies

Standardized mortality ratio.

at a hospital for orthopedic disorders. The positive findings reported in 1981 were unchanged with expansion of the study population (Trichopoulos, Kalandidi, and Sparrows, 1983).

The results of other subsequently reported case-control studies have also demonstrated statistically significant associations between involuntary smoking and lung cancer (Table 6.6). The findings from the more recent reports greatly strengthen the evidence from the earlier studies. Several of the newer studies included relatively large numbers of nonsmokers (Garfinkel, Auerbach, and Joubert 1985; Akiba, Kato, and Blot 1986; Dalager et al. 1986; Gao et al. 1987; Lam et al. 1987). Furthermore, in most of these studies, involuntary smoking was assessed in greater detail than in the earlier reports so that exposure-response relationships could be examined more fully.

The results of two other investigations have also been interpreted as showing an increased lung cancer risk associated with passive smoking although both have methodologic limitations. In Germany, Knoth, Bohn, and Schmidt (1983) described a series of fifty-nine female lung cancer cases of which thirty-nine were in nonsmokers. Based on census data, these investigators projected that a much greater than expected proportion of these nonsmokers had lived in households with smokers. This report did not include an appropriate comparison series, however. Gillis et al. (1984) described the preliminary results of a cohort study of 16,171 males and females in western Scotland; domestic exposure to tobacco smoke increased the lung cancer risk for nonsmoking men but not for women. The report was based on only sixteen cases of lung cancer in nonsmokers, however.

The results of some investigations indicate lesser or no effects of exposure to ETS (Table 6.6). In these studies, however, confidence limits for the relative risks associated with marriage to a smoker are wide and overlap with the confidence limits in the studies with significant results (NRC 1986a). Two separate case-control studies in Hong Kong, where lung cancer incidence rates in females are particularly high, did not indicate excess risk from passive smoking (Chan et al. 1979; Chan and Fung 1982; Koo, Ho, and Saw 1984; Koo, Ho, and Lee 1985; Koo et al. 1987). In the more recent of the two studies, the questionnaire comprehensively assessed cumulative exposure from home and workplace sources (Koo, Ho, and Saw 1984; Koo, Ho, and Lee 1985; Koo et al. 1987). A subsequent study in

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Hong Kong did find a significant association of spouse smoking and lung cancer risk (Lam et al. 1987). Lee and co-workers (Lee, Chamberlain, and Alderson 1986) reported a hospital-based case-control study in England. Although the investigators considered that their findings indicated little or no effect of involuntary smoking, the case series was small. Another recent hospital-based case-control study, conducted in Japan, also did not show an association between lung cancer risk and spouse smoking (Shimizu et al. 1988).

The results of the American Cancer Society's prospective cohort study of mortality in 176,139 nonsmoking women have also been construed by many as not showing an association between involuntary smoking and lung cancer (Garfinkel 1981). However, the standardized mortality ratios for the nonsmoking women with husbands who smoked were greater than unity, but not significantly greater. Recent and preliminary results from a nationwide case-control study also did not demonstrate increased lung cancer risk from domestic exposure to tobacco smoke (Kabat and Wynder 1984). In another case-control study that was performed in Los Angeles, Wu et al. (1985) did not find significantly increased risk for adenocarcinoma associated with involuntary smoking in smoking and nonsmoking women. These investigators estimated exposure from parental smoking, spouse smoking, and workplace sources. The relative risk for lung cancer was slightly, but not significantly, increased by exposure from spouse smoking and from smoking by co-workers.

The current extent of the data on involuntary smoking and lung cancer contrasts with the more extensive literature cited in the 1964 surgeon general's report, which characterized active cigarette smoking as a cause of lung cancer (U.S. Public Health Service [PHS] 1964). The variability of the data on involuntary smoking also contrasts with that on active smoking. However, most of the studies on involuntary smoking and lung cancer have small numbers of cases, and confidence intervals for the effect of involuntary smoking in the various studies would overlap. Variation in the results of the studies may also reflect random and nonrandom errors in the classification of exposure to ETS. In fact, assessment of exposure relevant to lung cancer appears more difficult than for other health effects of involuntary smoking. The relevant exposures may begin at birth and occur under a wide variety of circumstances. Thus, some inconsistency among the studies would be anticipated in the face of the relatively small study populations and the difficulties of estimating exposure.

In spite of the variable epidemiologic evidence, ETS has been characterized as a cause of lung cancer. The International Agency for Research on Cancer of the World Health Organization (IARC 1986) has concluded that "passive smoking gives rise to some risk of cancer." In its monograph on tobacco smoking, the agency supported this conclusion on the basis of the characteristics of sidestream and mainstream smoke, the absorption of tobacco smoke materials during involuntary smoking, and the nature of dose-response relationships for carcinogenesis.

The NRC (NRC 1986a) and the U.S. surgeon general (U.S. DHHS 1986) have also concluded that involuntary smoking increases the incidence of lung cancer in

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nonsmokers. In reaching this conclusion, the NRC (NRC 1986a) cited the biologic plausibility of the association between exposure to ETS and lung cancer and the supporting epidemiologic evidence. Based on a pooled analysis of the epidemiologic data which adjusted for bias, the report concluded that the best estimate for the excess risk of lung cancer in nonsmokers married to smokers was 25 percent. The 1986 report of the surgeon general (U.S. DHHS 1986) characterized involuntary smoking as a cause of lung cancer in nonsmokers. This conclusion was based on the extensive information already available on the carcinogenicity of active smoking, on the qualitative similarities between ETS and mainstream smoke, and on the epidemiologic data on involuntary smoking.

The extent of the lung cancer hazard associated with involuntary smoking in the United States remains uncertain, however (U.S. DHHS 1986; Weiss 1986). The epidemiologic studies provide varying and imprecise measures of risk, and exposures have not been characterized for large and representative population samples. Risk estimation procedures have been used to describe the lung cancer risk associated with involuntary smoking, but assumptions and simplifications must be made in order to use this method. For example, Repace and Lowrey (1985) calculated that approximately 5,000 lung cancer deaths occur annually in U.S. nonsmokers as a result of involuntary smoking. Wells (1988) attributed 3,000 lung cancer deaths annually in the United States to involuntary smoking.

OTHER CANCERS

Several recent reports have suggested that exposure to ETS may increase the risk of cancer at sites other than the lung. One study found that in children, maternal exposure to environmental tobacco smoke during pregnancy was associated with increased risk of brain tumors (Preston-Martin et al. 1982), and in another study paternal but not maternal smoking increased the risk of childhood rhabdomyosarcoma (Grufferman et al. 1982). Such effects might arise from smoking-induced changes in germ cells of the parents or through transplacental exposure rather than as a direct effect of smoke inhalation (Everson 1980; Grufferman et al. 1983).

In adults, involuntary smoking has been linked to a generally increased risk of malignancy and to excess risk at specific sites. Miller (1984) interviewed surviving relatives of 537 deceased nonsmoking women in western Pennsylvania concerning the smoking habits of their husbands. A significantly increased risk of cancer death (odds ratio = 1.94, $p < .05$) was found in women who were married to smokers and also not employed outside the homes. The large number of potential subjects who were not interviewed and the possibility of information bias detract from this report.

Sandler and colleagues (Sandler, Everson, and Wilcox 1985, Sandler et al. 1985; Sandler, Wilcox, and Everson 1985) conducted a case-control study on the effects of exposures to ETS during childhood and adulthood on the risk of cancer. The 518 cases included cancers of all types other than basal cell cancer of the skin; the cases and the matched controls were between the ages of fifteen and fifty-nine years. For all sites combined, significantly increased risk was found for parental smoking (crude

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odds ratio = 1.6) and for marriage to a smoking spouse (crude odds ratio = 1.5), the effects of these two exposures were independent (Sandler, Wilcox, and Everson 1985). Significant associations were also found for some individual sites: for childhood exposure (Sandler et al. 1985), maternal and paternal smoking increased the risk of hematopoietic malignancy, and for adulthood exposure (Sandler, Everson, and Wilcox 1985), spouse's smoking increased the risk for cancers of the female breast, female genital system, and the endocrine system. These findings are primarily hypothesis generating and require replication. In a case-control study such as reported by Sandler et al. (1985), biased information on exposure to environmental tobacco smoke is of particular concern. Hirayama (1984) has reported significantly increased mortality from nasal sinus cancers and from brain tumors in nonsmoking women married to smokers in the Japanese cohort. In a case-control study of bladder cancer, involuntary smoke exposure at home and at work did not increase risk (Kabat, Dieck, and Wynder 1986). Cervical cancer, which has been linked to active smoking, was associated with duration of involuntary smoking in a case-control study in Utah (Slattery et al. 1989).

These associations of involuntary smoking with cancer at diverse nonrespiratory sites cannot be supported readily with arguments for biologic plausibility. Increased risks at some of the sites, for example, cancer of the nasal sinus and female breast cancer, have not been observed in active smokers (U.S. DHHS 1982). In fact, the IARC has concluded that effects would not be produced in passive smokers which would not be produced to a larger extent in active smokers (IARC 1986).

CARDIOVASCULAR DISEASE

Although extensive data establish active cigarette smoking as a causal risk factor for cardiovascular diseases (U.S. DHHS 1983), few studies have examined involuntary smoking as a risk factor for these diseases. In the cohort of nonsmoking Japanese women, Hirayama (1983) found a small, statistically significant increased risk of death from ischemic heart disease associated with the husband's smoking. Garland et al. (1985) prospectively determined mortality from ischemic heart disease in nonsmoking older women residing in southern California. After adjustment for established risk factors, marriage to a smoking spouse was associated with a relative risk of 2.7 ($p < .10$). Gillis et al. (1984) assessed the baseline prevalence of cardiovascular symptoms and major electrocardiographic abnormalities in a population sample residing in Scotland and then determined cause-specific mortality for up to ten years of follow-up. In a preliminary report, involuntary smoking was not associated with the prevalence of cardiovascular symptoms at baseline or with cardiovascular mortality on follow-up. A case-control study in England did not show increased risk for ischemic heart disease or for stroke in nonsmokers married to smokers (Lee, Chamberlain, and Alderson 1986).

The effect of involuntary smoking was assessed among the nonsmoking male participants in the MRFIT; these men had been selected to be in the upper 10–15 percent of risk for mortality from coronary artery disease, based on a score from

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the Framingham study (Svendsen et al. 1987). In comparison with men married to nonsmokers, never smokers with smoking wives had increased risk for coronary heart disease (relative risk = 2.11, 95 percent confidence interval 0.69–6.46), fatal or nonfatal coronary heart disease event (relative risk = 1.48, 95 percent confidence interval 0.89–2.47), and death from any cause (relative risk = 1.96, 95 percent confidence interval 0.93–4.11). These relative risks changed little when former smokers were included in the analysis or with adjustment for other risk factors for coronary heart disease.

Helsing and colleagues (1988) reported on the heart disease mortality of nonsmokers enrolled in a cohort study in Washington County, Maryland. In comparison with persons married to nonsmokers, both men and women married to smokers had a significantly increased risk of dying from heart disease (*RR* = 1.3 for males and 1.2 for females).

The effects of involuntary exposure to tobacco smoke on athletic performance, which largely depends on cardiovascular responses, have also been examined. Such effects derive plausibility from the composition of ETS, which contains agents that might affect level of lung function and oxygen delivery by the cardiovascular system. Irritation of the upper and lower respiratory tracts following acute exposure might also adversely affect performance when exercise requires high minute ventilation.

Of the many toxic agents in ETS, CO and nicotine are candidates to influence performance adversely because of their cardiovascular effects (Schievebein and Richter 1984). CO interferes with oxygen transport by avidly binding to hemoglobin to form carboxyhemoglobin and by shifting the oxyhemoglobin dissociation curve to the left. Nicotine releases epinephrine and norepinephrine and elevates pulse rate, blood pressure, and oxygen demand. Although these effects of CO and nicotine might impair performance, exposures to ETS are generally at concentrations below which physiologic effects would be expected to occur (Schievebein and Richter 1984; U.S. DHHS 1986).

In two studies, healthy nonsmoking subjects were exposed to tobacco smoke and their performance subsequently assessed (Pimm, Silverman, and Shephard 1978; McMurray, Hicks, and Thompson 1985). In both studies, attempts were made to blind the subjects as to whether clean air or smoke-contaminated air was inhaled. Given the unmistakable odor and other characteristics of cigarette smoke, it seems unlikely that these studies were truly blinded, and their findings must be interpreted with this constraint.

Pimm, Silverman, and Shephard (1978) exposed ten males and ten females to levels of cigarette smoke described as characteristic of "levels typically encountered in public buildings." The subjects were exposed in a chamber and subsequently underwent a progressive exercise test. Maximal oxygen uptake was not significantly affected, and a consistent pattern of adverse effects on lung function was not found. The subjects did have symptomatic responses to the exposure.

In the study conducted by McMurray, Hicks, and Thompson (1985), air or cigarette smoke was inhaled by eight female subjects through a mouthpiece, and

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submaximal and maximal exercise performance were assessed. The inhalation of cigarette smoke led to a reduction of maximal oxygen uptake and a reduction of time to exhaustion. Effects on the respiratory exchange ratio (R), maximal blood lactate, and ratings of perceived exertion were also found during the maximal testing. Adverse effects of smoke exposure were also suggested by the submaximal testing.

TOTAL MORTALITY

Several cohort studies provide information on involuntary smoking and mortality from all causes. In the Scottish cohort study, total mortality was increased for women living with a smoker but not for men (Gillis et al. 1984). As described previously, total mortality was also increased among nonsmoking participants in MRFIT who lived with smokers (Svendsen et al. 1987). In contrast, mortality was not increased for nonsmoking female subjects in a study in Amsterdam (Vandenbroucke et al. 1984). Neither the study in Scotland nor the study in Amsterdam controlled for other factors that influence total mortality. In the cohort study in Washington County, all-cause mortality rates were significantly increased for men ($RR = 1.17$) and for women ($RR = 1.15$) after adjustment for housing quality, schooling, and marital status (Sandler et al. 1989).

ODOR AND IRRITATION

Tobacco smoke contains numerous irritants, including particulate material and gases (U.S. DHHS 1986). Both questionnaire surveys and laboratory studies involving exposure to ETS have shown annoyance and irritation of the eyes and upper and lower airways from involuntary smoking. In several surveys of nonsmokers, complaints about tobacco smoke at work and in public places were common (U.S. DHHS 1986): about 50 percent of respondents complained about tobacco smoke at work, and a majority was disturbed by tobacco smoke in restaurants. The experimental studies show that the rate of eye blinking is increased by ETS, as are complaints of nose and throat irritation (U.S. DHHS 1986). In the study of passive smoking on commercial airline flights reported by Mattson and colleagues (1989), changes in nose and eye symptoms were associated with nicotine exposure. The odor and irritation associated with ETS merit special consideration because a high proportion of nonsmokers are annoyed by exposure to ETS, and control of the concentrations in indoor air poses difficult problems in the management of heating, ventilating, and air-conditioning systems.

EXPERIMENTAL STUDIES

Experimental exposure of volunteer subjects has provided convincing evidence of the irritant effects of exposure to ETS. Studies reviewed in the 1986 report of the surgeon general (U.S. DHHS 1986) documented eye irritation through both objective measurements of blink rates and of stability of the precorneal tear film and subjective reports. Symptoms of nose and throat irritation were also associated

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with ETS exposure. More recent experimental studies have provided further data concerning the odor and annoyance associated with ETS exposure.

The usual experimental setting to assess the effects of indoor contaminants on the senses is exposure in a climate-controlled environmental chamber with relatively inert surfaces, for example, aluminum or stainless steel, and variable ventilation. To investigate occupancy odor, human beings occupy the chamber for a given time. Judges of the odor of interest either enter the chamber briefly or place their faces into a box fed with the atmosphere of the chamber and may thus be considered to visit the space. The odor judgment may comprise a mark on an annotated rating scale (e.g., *no odor* to *overpowering odor*) or the choice of a matching odor intensity, generally using a device called an *olfactometer*, which delivers the vapor of a standard odorant, such as *n*-butyl alcohol (1-butanol), at various concentrations.

Many modern investigations also obtain judgments of acceptability in order to "calibrate" intensity judgments. Acceptability judgments address the question: How many people object to any given level of odor (or irritation)? The answer depends upon individual differences in olfactory sensitivity and upon aesthetic criteria. Whereas we can expect average intensity judgments to remain constant through the decades for any fixed stimulus, we can expect acceptability judgments to shift with prevailing standards.

Figure 6.1 shows the relationship between ventilation rate and occupant satisfaction in a recent study conducted in a climate chamber with from four to twelve occupants (Cain et al. 1983). Visitors made judgments of air circulated through an outside sampling box and were therefore naive to the conditions of occupancy. The scale refers to the concentration of 1-butanol matched to the occupancy odor present after one hour of occupancy. Both odor level and dissatisfaction decreased with increases in ventilation rate.

The ventilation standard of the American Society of Heating, Refrigerating, and Air-Conditioning Engineers (ASHRAE) (1989) recommends a maximum of 20 percent dissatisfaction among visitors to a space. By this criterion, the data from the investigation imply the need for 17 cubic feet per minute (cfm) per occupant (Figure 6.1). The ASHRAE standard suggests 15 cfm or more per occupant for most spaces, for example, 15 cfm for classrooms, libraries, auditoriums, and dormitories; 20 cfm for offices, conference rooms, dining rooms, and lobbies; 25 cfm for discotheques and beauty shops; 30 cfm for bars and casinos; and 60 cfm for smoking lounges. Thus, the ASHRAE standard specifies sufficient air to control odor associated with the occupants of a space.

When cigarettes were smoked in the climate chamber, odor level increased markedly (Figure 6.2). Figure 6.2 shows ETS odor for various conditions of smoking: intermittent (four cigarettes per hour) or continuous (eight and sixteen cigarettes per hour). The intensity of the odor increased with the number of cigarettes smoked and declined with increasing ventilation. As shown in Figure 6.3, the degree of dissatisfaction mirrored the higher odor level. Based on the rule of 20 percent maximum dissatisfaction, the ventilation rate required per cigarette

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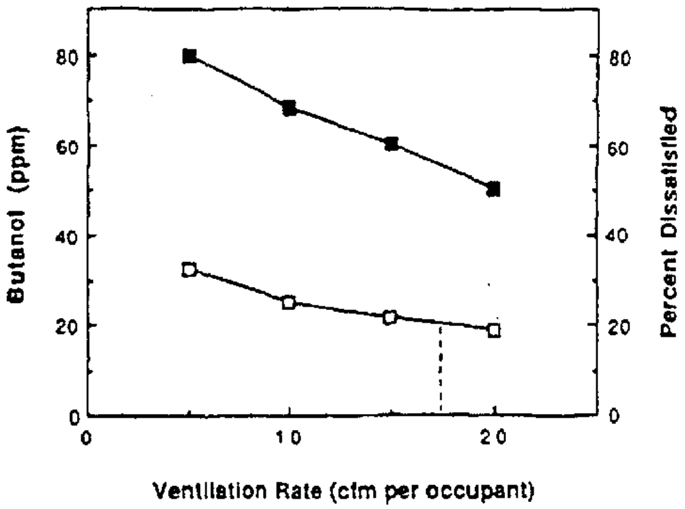

Figure 6.1. Relation between level of occupancy odor (indicated by concentration of 1-butanol matched to the odor [*filled squares*]) and ventilation rate per occupant when four to twelve persons occupied a climate chamber for an hour. Judgments of odor were made by visitors who sampled the air of the chamber at a remote sampling box. Also shown is the frequency of dissatisfaction expressed by the visitors in response to the question, "Is the air acceptable or unacceptable (*unfilled squares*)?" The *dashed line* shows the ventilation rate that led to 20 percent dissatisfaction. *Source:* Reprinted with permission from *Atmospheric Environment* 17, Cain W. S. et al., Ventilation requirements in buildings—1. Control of occupancy odor and tobacco smoke odor. Copyright 1983, Pergamon Press PLC.

during active smoking exceeded 4,000 ft^3. In order to convert ventilation per cigarette into ventilation rate per person for typical conditions of occupancy in a "smoking-permitted" space, we assumed that 10 percent of the occupants would be smoking at any given time. The resulting requirement for ventilation was 53 cfm, more than three times that for nonsmoking occupancy.

The higher ventilation rate for smoking calculated from these data does not appear to represent a special aversion to the odor of cigarettes on the part of the judges. The judges, one-third of whom were smokers and two-thirds of whom were not, seemed to base their dissatisfaction strictly on odor intensity. Degree of dissatisfaction varied with odor intensity in the same way for both occupancy odor and tobacco smoke odor (Figure 6.4): stronger odors yielded greater dissatisfaction regardless of the source of odor.

The adequacy of the ASHRAE standard can be evaluated using these experimental data. The standard recommends 60 cfm per occupant in a smoking lounge, where presumably most or all occupants may be smoking. If 100 percent rather than 10 percent of the occupants smoke simultaneously, then the ventilation rate would need to exceed an unachievable 500 cfm per occupant. If 50 percent smoke

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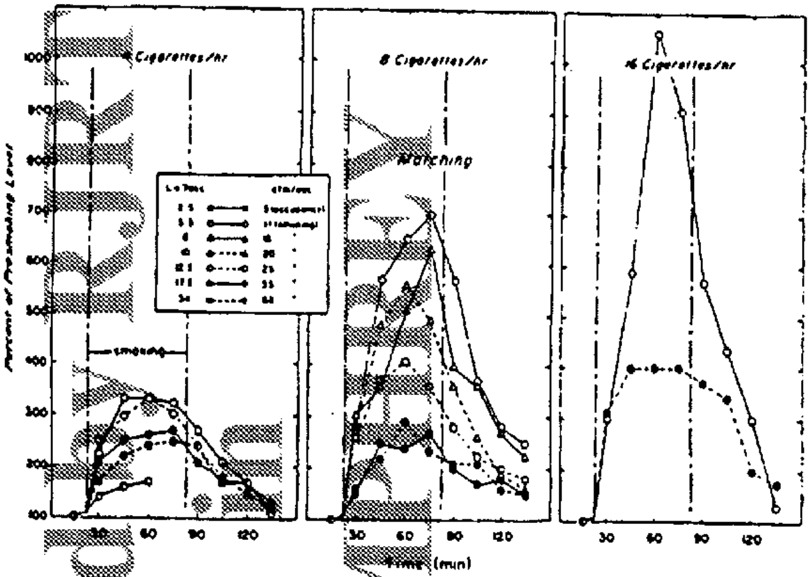

Figure [illegible]. Intensity of ETS odor perceived by visitors to the sampling box during and after intermittent (four cigarettes per hour) or continuous (eight to sixteen cigarettes per hour) smoking in the climate chamber. Results are expressed relative to level of butanol matched to odor during presmoking occupancy. The *open squares* in the *left panel* show a function for nonsmoking occupancy for comparison. *Ventilation rate per occupant under smoking conditions* refers to smokers, who were the only occupants in the chamber. *Source:* Reprinted with permission from *Atmospheric Environment* 17, Cain W. S. et al., Ventilation requirements in buildings—I. Control of occupancy odor and tobacco smoke odor. Copyright 1983, Pergamon Press PLC.

simultaneously, perhaps a more realistic scenario, then the rate would need to exceed a still unachievable 250 cfm per occupant. The maximum achievable rate for typical design occupancy in a mechanically ventilated space will usually equal about 60 cfm per occupant, although a generous allotment of space per person can increase that value.

The smoker, however, seems less concerned about the odor of ETS than the nonsmoker. Smokers as a group seem satisfied with about one-quarter of the ventilation air needed by a group containing a typical proportion of smokers and nonsmokers. Hence, a rate of 60 cfm per occupant in smoking areas may almost meet the customary ASHRAE criterion of a maximum of 20 percent dissatisfaction for a group of smokers. The data from the investigation suggest that under typical conditions of smoking occupancy (10 percent smoking at any given time), nonsmokers would need over 100 cfm per occupant to hold dissatisfaction at only 20 percent. We do not know whether the difference between smokers and nonsmokers reflects olfactory sensitivity to ETS or other criteria.

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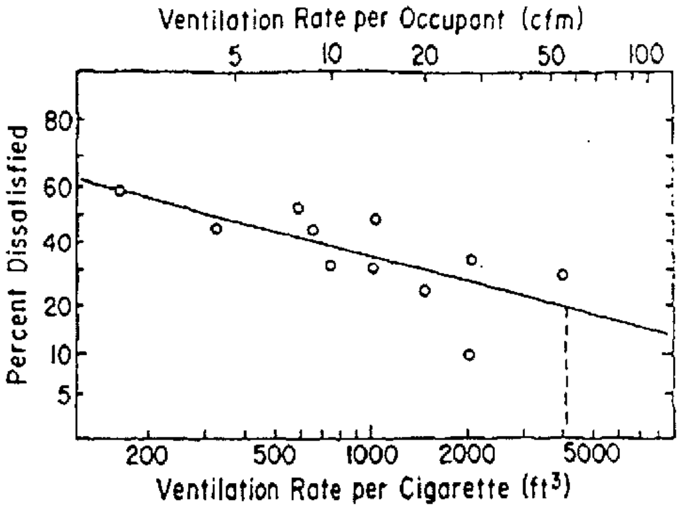

Figure 6.3 Percent dissatisfaction among visitors versus ventilation during the last 15 minutes of smoking in the experiments shown in Figure 6.2. The ventilation rate per cigarette is based on 7.5-minute smoking time per cigarette. The ventilation rate per occupant is adjusted to typical conditions of smoking occupancy in which 10 percent of occupants will be smoking at any given time. *Source* Reprinted with permission from *Atmospheric Environment* 17, Cain W. S. et al., Ventilation requirements in buildings—I. Control of occupancy odor and tobacco smoke odor. Copyright 1983, Pergamon Press PLC.

Clausen and co-workers (1986b) found similar differences in tolerance of ETS odor between smokers and nonsmokers. For any given level of odor (expressed as concentration of butanol), nonsmokers expressed much more dissatisfaction than smokers. For both groups, odor intensity and dissatisfaction were associated, but the relationship was steeper for nonsmokers. At the point at which 20 percent of the smokers expressed dissatisfaction, almost half of nonsmokers were dissatisfied with the air quality.

In a laboratory situation in which other sources of combustion can be eliminated, CO can serve as an index of the level of ETS. Clausen related dissatisfaction to concentration of CO in ETS as well as to matched level of butanol; in the experiment, odor intensity and the increment in CO concentration due to smoking were strongly correlated. The concentration at which 20 percent of the nonsmokers expressed dissatisfaction fell about eight times below that at which 20 percent of the smokers expressed dissatisfaction.

As ETS enters the atmosphere, its many chemical constituents react both chemically and physically with each other and with surrounding materials. Nevertheless, the odor of ETS behaves over short time periods as though ETS were a stable contaminant. After the source has been removed, ETS odor decays in a manner

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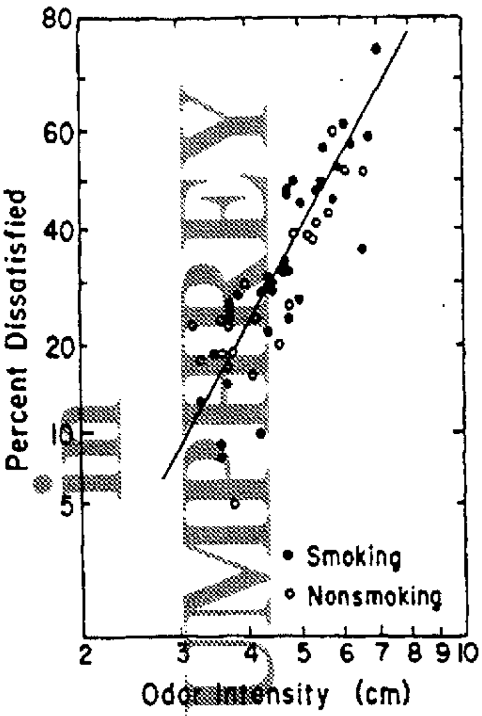

Figure 6.4 Percent dissatisfaction versus odor intensity (graphic rating) during smoking and nonsmoking conditions. *Source:* Reprinted with permission from *Atmospheric Environment* 17, Cain W. S. et al., Ventilation requirements in buildings—I. Control of occupancy odor and tobacco smoke odor, Copyright 1983, Pergamon Press PLC.

entirely predictable from ventilation rate (Clausen et al. 1985). In this respect, it differs from occupancy odor, which has a half-life of 55 minutes, presumably dictated by slow oxidation of chemical constituents into less odorous products (Clausen et al. 1986a).

Standards for ventilation have focused on the reactions of the visitor to a space, rather than those of the occupant, on the assumption that the visitor will be more sensitive to, and hence more critical of, the contaminant than the person who has adapted to it. On the other hand, a focus on the visitor may not adequately address irritation, which is an important time-dependent sensory response of the occupant. Whereas air containing an irritant may initially be only slightly irritating, it may become intolerably so over time.

Figure 6.5 illustrates the time course of eye irritation experienced by occupants

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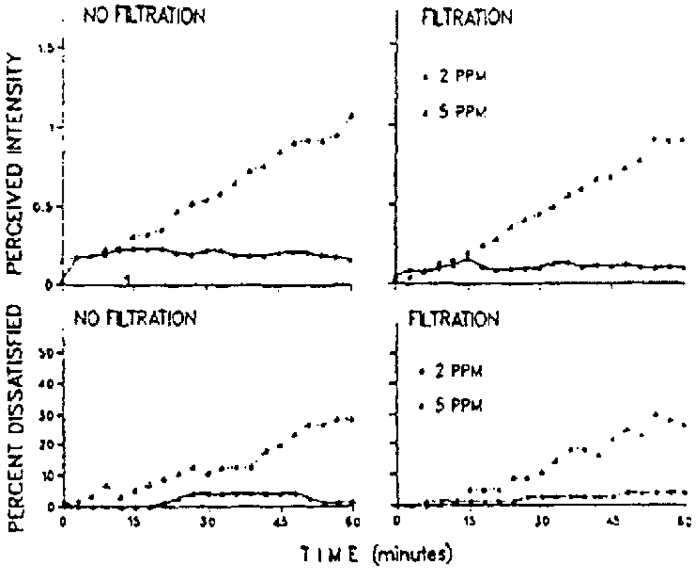

Figure 6.5. Perceived magnitude of eye irritation and degree of dissatisfaction expressed by occupants exposed to ETS for an hour. Concentrations of CO were held constant throughout the exposures and indicate severity of exposure. *Filtration* refers to elimination of particles via electrostatic precipitation. Filtration had little effect on irritation. *Source:* Reprinted with permission from *Atmospheric Environment* 21. Cain W. S. et al., Environmental tobacco smoke: Sensory reactions of occupants. Copyright 1987, Pergamon Press PLC.

exposed to ETS at constant concentrations of 2 or 5 ppm of CO, used as an index of ETS concentration (Cain et al. 1987). The lower concentration led to slight, although statistically significant, irritation above the presmoking baseline. The higher concentration led to irritation that increased over time in sensory magnitude and caused an increasing degree of dissatisfaction. Whereas essentially none of the occupants found the irritation objectionable at first, by the end of an hour about 30 percent were irritated. In an extension of this study, Clausen, Mäller, and Fanger (1987) found that a level of 20 percent dissatisfaction would occur at a concentration of 3.8 ppm of CO. A comparison with the odor judgments of visitors suggests that only smokers would find such a level tolerable by the "20 percent rule." Clausen, Mäller, and Fanger (1987) estimated that the ventilation rate necessary to control irritation of occupants to a dissatisfaction level of 20 percent would equal only one-tenth of that needed to control odor perceived by visitors to the same level of dissatisfaction.

Although Clausen and co-workers did not argue in favor of lowering ventilation to meet only the dissatisfaction of occupants, others have suggested this approach

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(Winneke et al. 1984). Cain and co-workers (1987) cautioned against handling irritation and odor in the same fashion. The appropriateness of applying the 20 percent rule to dissatisfaction based on irritation alone, as it is applied to dissatisfaction based on odor alone, has been questioned. Whereas odor may be interpretable narrowly on grounds of comfort, irritation would seem interpretable on grounds of health.

CONTROL

The standards of ASHRAE mandate specific levels of ventilation to provide occupant satisfaction with indoor air quality. The experimental studies reviewed above provide insights concerning the level of ventilation needed to achieve satisfaction for nonsmokers and for smokers in the presence of ETS. In actual indoor environments, when ventilation fails to eliminate the contaminant entirely, the physical interaction of ETS with surfaces may increase the need for ventilation. Because the liquid aerosol of ETS, known in the aggregate as *tar*, adsorbs strongly to walls, fabrics, and so on, it becomes a source of odor as components volatilize to the air. The background odor of the emitted products carries its own demands for ventilation, predictable in part from the typical amount of smoking in a space (Clausen et al. 1986b).

Obviously, ETS can be readily controlled by eliminating smoking from indoor environments or its impact lessened by limiting smoking to specific areas. Air cleaning offers another approach.

Although the odor of ETS can be postulated to come from its vapor phase and the irritation from its particulate phase, recent investigations that have employed electrostatic air cleaning have shown clearly that the gas phase accounts for most of the odor and irritation (Hugod 1984; Weber 1984). Comparison of the right and left sides of Figure 6.5 shows that elimination of the particulate phase had only a trivial effect on the eye irritation caused by ETS at 2 and 5 ppm carbon monoxide (Cain et al. 1987). The findings were similar for judgments of odor and of nose and throat irritation. Clausen, Mäller, and Fanger (1987) confirmed these results. In finding that particles played essentially no role in odor, both investigations also confirmed Clausen and colleagues' (1985) earlier experiments with visitors. Particle filtration holds no promise for immediate elimination of the discomfort of exposure to ETS. The major advantage of such air cleaning derives from reduction of haze and collection of tar that would otherwise adsorb elsewhere in the space.

Although both the odor and irritation of ETS come from the vapor phase, the chemical constituents responsible for these two effects may be different. Undoubtedly, the odor comes from a very large number of constituents. The sense of smell responds to almost all airborne organic materials present in sufficient concentration (Cain 1988). For one substance, however, a "sufficient concentration" may fall a millionfold below that of another. Furthermore, individual constituents may combine perceptually in mixtures in complicated, nonlinear ways.

Many single compounds can cause irritation at the concentrations present in ETS, which represents a complex mixture with numerous components capable of

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causing odor and irritation. Little is known about the effects upon perception of the combination of ETS irritants although it is known that odor and irritation interact (Cain and Murphy 1980). Irritation can suppress the perception of odor and vice versa (Cain, See, and Tosun 1986). Insofar as irritation may have a less complex origin than odor, it may offer easier opportunities for control through filtration. As yet, however, experiments on ETS have told more about what fails to cause irritation than about what causes it (Weber, Jermini, and Grandjean 1976; Weber-Tschopp, Fischer, and Grandjean 1977; Weber-Tschopp et al. 1977; Hugod, Hawkins, and Astrup 1978).

The complexity of ETS more or less assures that almost any means of air cleaning will eliminate part of it even though no simple procedure will eliminate all of it. Through the use of air washing that presumably eliminated some water-soluble constituents of ETS, Clausen, Møller, and Fanger (1987) achieved some reduction in level of dissatisfaction although not in the perceived intensity of ETS. The results offered little encouragement for using air washing alone but showed that the odor character of ETS can play some role in the degree of acceptance.

A combination of particulate air cleaning and vapor phase cleaning via adsorption on activated carbon or via chemisorption on oxidant-impregnated alumina can likely control both the irritation and odor of ETS to some degree. Unfortunately, standards for assessing the efficacy of vapor phase filtration media have not been developed. The installation of such media occurs more commonly in special environments, for example, libraries and computer facilities, under expert guidance than in spaces designed for general occupancy. In the overwhelming majority of cases, attempts to control ETS rely on ventilation (dilution). As shown by the experimental studies, however, ventilation has limitations.

SUMMARY

The effects of active smoking and the toxicology of cigarette smoking have been examined comprehensively. The periodic reports of the U.S. surgeon general and other summary reports have considered the extensive evidence on active smoking; these reports have provided definitive conclusions concerning the adverse effects of active smoking, which have prompted public policies and scientific research directed at prevention and cessation of smoking.

Although the evidence on involuntary smoking is not as extensive as that on active smoking, health risks of involuntary smoking have been identified. The 1986 report of the U.S. surgeon general (U.S. DHHS 1986) and the 1986 report of the NRC (NRC 1986a) both concluded that involuntary exposure to tobacco smoke increases respiratory infections in children, increases the prevalence of respiratory symptoms in children, reduces the rate of functional growth as the lung matures, and causes lung cancer in nonsmokers. Involuntary smoking may also adversely affect asthmatics, and annoyance and irritation are well documented. These adverse effects of involuntary exposure to tobacco smoke have provided a strong

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rationale for policies directed at reducing and eliminating exposure of nonsmokers to ETS (U.S. DHHS 1986). Complete protection of nonsmokers in public locations and the workplace may require the banning of smoking since the 1986 report of the surgeon general (U.S. DHHS 1986) concluded that "the simple separation of smokers and nonsmokers within the same air space may reduce, but does not eliminate, the exposure of nonsmokers to environmental tobacco smoke."

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EXECUTIVE SUMMARY as of May 21, 1994

AIRCRAFT CABIN ENVIRONMENTAL SURVEY

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There is a general perception among many passengers and flight crew that flying on commercial aircraft is unhealthy. Respiratory complaints are among the most common. For many of us, the prospect of being confined to a small noisy space (≈2 m^3/person) and breathing dry, conditioned air at pressures comparable to that at altitudes up to 8000 feet is something we endure as a necessity of modern travel. Most of us, unaware of the real and documented hazards of increased exposure to ozone and radiation, express concern about the also real possibility of airborne infection. Knowledge that the new generation of aircraft have recirculated air to reduce the cost of providing 100% outdoor air ventilation has only heightened those concerns. In recent years, professional engineering societies have revised ventilation standards towards increasing fresh air in recognition of dissatisfaction with previous codes. On the other hand, aircraft designs have actually reduced the fresh air delivery capacities of the environmental control units (ECUs). Passengers and cabin crew on a plane nearly full to capacity receive less than half the outside air per minute per person recommended for offices and schools.

Reduced amounts of outdoor air do not necessarily translate to poor air quality and increased risk of disease. Air cleaning and removal of pollutant sources mitigate some of the effects of decreasing dilution air. Even with recirculating ventilation systems oxygen is not depleted, nor does carbon dioxide increase to levels that interfere with respiration. Of concern, however, is the adequacy of the strategies used (i.e., recirculation and filtration) to offset the effects on air quality of reducing the amount of outdoor air produced. The effectiveness of filtration is based on the efficiency with which relevant particles and gases are removed from the recirculated air. This efficiency is determined by the type of filters, air flow through filters, and by the pattern and rate of air movement in the cabin. However, the practical effectiveness of the ventilation system is ultimately determined by the human response and, therefore, includes additional characteristics of the cabin environment, such as air mixing, direction of bulk air flow, temperature, numidity, draftiness, turbulence, and odors in addition to actual contaminant levels.

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Using a hand-held portable dust collector fitted with a cloth collection bag, dust from the carpet and seat was collected on 16 flight segments. Samples were collected over approximately 4-5 minutes and yielded up to 2 grams of dust. Large particles were removed by sifting the dust through a 425 μm mesh sieve. Portions of this dust were sent to VESPA Labs for cat and mite antigen testing. A portion was retained for fungal and endotoxin testing. Fungi were recovered by suspending the dust in an aqueous solution and spread plating the suspension onto malt extract agar (MEA) and incubating at room temperature for 5 to 7 days. Fungi were identified to genus and reported as fungal CFUs per gram of dust. Endotoxin levels in 6 dust samples were analyzed by a Limulus-based method developed for analysis of environmental samples. Details of laboratory procedures are reported elsewhere.

During the cruise period of six flights, 2-hour long integrated air samples for VOC analysis were collected using Summa stainless steel canisters. Canisters were sent to Performance Analytical, Inc. where they were analyzed according to EPA's TO-14 GC/MS for 55 volatile organic compounds. Using a GC/FID, 50 compounds were examined which were considered to be ozone precursors in urban air. An additional 15 compounds were quantified.

In a related survey, we have deployed passive ozone badges (Koutrakis et al. *Analytical Chemistry* 65:209-214; 1993) on close to 100 trans-Pacific flights. The study protocol varied over the course of the year-long investigation, providing information on integrated ozone concentrations over the duration of the flight (up to 15 hours per flight) and for periods as short as 3 hours. The three-hour segments were when the aircraft was flying over the Aleutian Islands. On several flights, up to four locations in the plane were monitored. The badges were analyzed by ion chromatography in Harvard laboratories according to established protocols.

RESULTS

During cruise, average carbon dioxide levels on flights with partially recirculated air were up to twice the levels measured on planes with 100% outdoor air systems. CO_2 averaged 1500 ppm during flights with recirculation indicating that each passenger was receiving 8-10 cfm outdoor air. During the flight on which we operated two CO_2 monitors, the CO_2 concentrations in the rear of the coach were higher, suggesting a front to rear movement of air. An additional 6 flights will be examined to see if this is a consistent finding. During boarding and deboarding CO_2 was substantially higher than during cruise, with levels of 2000 ppm to 2500 ppm being typical. The boarding and pre-takeoff process varied in duration but usually lasted 30 minutes $\pm$ 15 minutes. In addition to CO_2, both relative humidity and temperature increased during boarding and deboarding. Conditions improved when the ECUs started to operate. Temperatures were comfortable, $73° \pm 2°F$, during flight but, as was to be expected, the air was quite dry with relative humidities in the range of 10% to 20%. Planes with recirculating

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All dust samples had measurable quantities of cat and mite antigen. Der pI, the common mite antigen of the Northern U.S., ranged between 0.1 and 1.9 µg/g. Fungal levels in dust were within the range commonly found in homes. Yeasts were more abundant in aircraft dust than is usual for other public environments. Endotoxin concentrations were recovered from dust in levels ranging from 150 to 450 EU/mg. These levels are within the upper range for house dust, and the mid range for cotton dust, a known source for disease-causing endotoxin aerosols.

DISCUSSION

Conclusions from this survey should be considered preliminary, and not comprehensive. For example, we did not sample for radiation or pesticides. A recent study by Wilson et al. (*Health Phy* 66[5]:493-502; 1994) reported annual radiation dose to domestic crew members between 1.0 and 1.8 mSv which has increased in the ten years since the previous survey. They calculate that the international crew might receive up to 3.8 mSv annually. We would expect the crew of North American carriers would receive more radiation than Australian crews because of latitudinal differences in routes.

Further, neither our survey nor any other study that we have found has adequately characterized exposure to infectious agents (viruses, TB, etc.) Sampling for infectious agents is problematic because of variability in sources, the low level of exposure necessary to cause disease, and difficulty with sampling methods. Nevertheless, given the recent Centers for Disease Control findings on a tuberculosis case study and other reports about transmission of infectious viruses on aircraft (Moser et al. *Am J Epidemiol* 110:1-6) and air-conditioned barracks (Brundage et al., *JAMA* 259[14]:2108-2112), it is important to pursue this issue further.

We can conclude that ASHRAE 62-89 ventilation standards for offices are not being met in airplanes with recirculating ECUs. This does not necessarily imply adverse conditions. Filtration can effectively mitigate some potential exposures and source control may be effective for agents that are not shed from passengers and crew. However, we found that the lowest outdoor air amounts were during the period of boarding. The auxiliary power units and ground ventilation systems appear to be insufficient to adequately ventilate planes. It is at these times that people are most likely to be actively stirring up dust and shedding bacteria, spores, and allergens from clothing and skin.

None of the volatile organic compounds (VOCs) found on-board the airplanes in this limited study were in sufficiently high concentrations individually to explain reported problems. The concentrations of total non-methane hydrocarbons is quite high and exceeds the concentrations associated with sick building complaints of eye and mucosal irritation. The mixture of VOCs is quite different on planes than reported for other environments with ethyl alcohol being the single most abundant compound. The mixture

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In our separate ozone survey, we found that, although the aircraft flying the Pacific routes were theorteically equipped with ozone destroying catalytic filters, our results clearly show the presence of elevated ozone. A substantial fraction of the flights, between 10% and 20%, had either the three-hour or flight-integrated ozone levels exceeding 100 ppb. Prolonged exposure to ozone at these levels has been shown to reduce lung function and cause eye and airway irritation. Human data suggesting chronic effects of ozone is insufficient to say with certainty that permanent damage is occurring. However, tests with animals show that chronic ozone exposure results in tissue damage, loss of elastic recoil, and increased collagen deposition at the bronchiolar level.

CONCLUSION

Some flight attendants and passengers complain of symptoms associated with sick buildings syndrome as well as cases of infectious disease assumed to be acquired on flights. Our survey combined with a previous DOT survey and data from the literature have provided some possible explanations for some symptoms. However, additional work is needed (1) to discover the actual rate of discomfort and disease resulting from air travel, (2) to document exposure (with air samples) to dust-borne endotoxin, and (3) to evaluate efficacy of remedial measures (e.g. improved cabin maintenance) that might be instigated.

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AIRLINER CABIN AIR QUALITY

(103-61)

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HEARING

BEFORE THE

SUBCOMMITTEE ON AVIATION

OF THE

COMMITTEE ON
PUBLIC WORKS AND TRANSPORTATION
HOUSE OF REPRESENTATIVES

ONE HUNDRED THIRD CONGRESS

SECOND SESSION

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Mr. OBERSTAR. The subcommittee will resume its hearing.

We welcome Dr. John Spengler, Harvard University. Mr. Paul Halfpenny, member of the Committee on Space Station Freedom. Dr. James Cone, Department of Medicine, University of California. And Ms. Sue Ludwig, flight attendant.

Welcome to all of you. Thank you for your patience. We begin with Dr. Spengler.

TESTIMONY OF DR. JOHN SPENGLER, DEPARTMENT OF ENVIRONMENTAL HEALTH, HARVARD UNIVERSITY, SCHOOL OF PUBLIC HEALTH, ACCOMPANIED BY DR. HARRIET BURGE, PROFESSOR OF MICROBIOLOGY, HARVARD UNIVERSITY; PAUL HALFPENNY, MEMBER, COMMITTEE ON SPACE STATION FREEDOM, DR. JAMES E. CONE, MEDICAL CONSULTANT, ASSOCIATION OF FLIGHT ATTENDANTS; SUE LUDWIG, FLIGHT ATTENDANT

Dr. SPENGLER. Thank you, Mr. Chairman.

On my right and left are two of my co-committee members from the National Academy of Science Committee on Aircraft Air Quality and Safety, where we delivered our report in 1986.

It is almost like what Yogi Berra said, it is deja vu all over again, because we are finding out that many of the recommendations we struggled with and thought very thoroughly about are still before us. They are still not being implemented. I think we are even in a better position with regard to empirical data to come back to underscore many of those recommendations.

I am Professor of Environmental Health at the Harvard School of Public Health, and I am accompanied by Dr. Harriet Burge, Professor of Microbiology also of Harvard. We continue to have an interest in air quality on airplanes, and even after our efforts on the National Academy committee, we continued to do our own surveys looking at particles, ozone, carbon dioxide, temperature, humidity, and radiation aboard commercial aircraft.

In fact, Sue Ludwig will talk about a year-long study we participated in and our laboratory supported which investigated ozone in aircraft cabins on transpacific flights. This is very important data you should take a look at.

In November of 1993, Dr. Timothy Johnson, the medical reporter for ABC News, asked for our assistance. For that effort we were given independence to design and conduct a survey on commercial aircraft, which we did between March and April of this year. The executive summary has been provided; which gives our findings and recommendations to date.

We are not going to discuss our results in detail as it is already on the docket. But we would like to answer questions that our survey may bring to mind among the Committee Members. In a few minutes I will highlight some of our points of view and recommendations.

Despite the prevailing assumption among the flight crew and passengers that they are at risk from illness, reproductive problems, and from cancer, we still do not have a thorough study of prevalence. The NIOSH birth outcome study of female flight attendants is a very important effort, but it will still be three or four years before the results of that study are in.

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Lung function changes are physiological miracles that can be studied at a much greater pace, so the results could be available much sooner. This needs to be done because we don't know the frequencies of illnesses. And case-control studies of cancer among the cabin crew and the pilots are needed desperately.

I just want to point out that radiation has taken on a new significance. There is a recent article published by Wilson, et al. in Health Physics this month about a study which looked at radiation exposures to the pilots and crew of Australian Airlines. They indicated that the exposures have increased over the previous 10 years. Also the exposures are higher in the cockpit than in the cabin of the airplane. By extrapolation, and because of the kinds of routes that American crews fly, the radiation exposures of U.S. crews could be higher than their Australian counterparts. They didn't draw that conclusion from their results, but I do. They also go on to say that the exposures could be five times the recommended level for the general public, which is one milli-sievert (MSV) per year averaged over five years. They strongly recommend that the occupational exposures of 20 milli-sievert (20 MSV) per year are not appropriate for this occupational group. They recommend something like 5 milli-sievert (5 MSV) per year. Even at 5 MSR per year, the risk of cancer is one in 4,000, which is actually higher than what we require for Superfund cleanups.

I want to do this as a duo and have Dr. Burge talk and amplify some of these points. I will come back then with a few simple things that the airlines could do and the manufacturers could do.

Dr. BURGE. Thank you.

I think it is impossible to deny at this point that a problem exists as far as air quality in aircraft, otherwise we wouldn't be sitting here. Many of the complaints that we are hearing or that I have heard today that stimulated our survey are similar to those that occur in office buildings (sick building syndrome and building-related illness). These are conditions that have been shown, at the very least, to reduce worker performance in office buildings.

There is some epidemiologic evidence for correlations between specific measures of air pollutants in office buildings and some of the symptoms we have been hearing about. For example, levels of carbon dioxide in excess of 800 parts per million are, in fact, correlated with symptoms of increasing complaints of sick building syndrome.

There is some evidence for a correlation between the presence of organic dust in office environments and symptoms of sick building syndrome. There certainly is strong evidence, as well as a good theoretical basis for an increase in infectious disease transmission in crowded environments with reduced fresh air ventilation.

The OSHA proposed regulations are specifically designed to address some of these issues. There is clear evidence in airplanes that, for example, carbon dioxide levels are well within the range in which sick building syndrome symptoms occur. In our study, as well as in the ATA studies and the GEOMET studies, carbon dioxide levels were significantly elevated. We are not saying carbon dioxide itself causes complaints, but it is a good indication in office buildings of increasing levels of health complaints.

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There is evidence in aircraft for infectious disease transmiss[ion] and, as I said earlier, there is good theoretical evidence to indicat[e] that increased ventilation will reduce the risk of transmission of infectious disease. For all three of the aircraft cabin air quality studies that we have heard discussed today, the data is really very similar. It is the interpretation (that is, what the data is considered to mean) that differs. There are two main reasons for these differences in interpretation:

Number one: We have heard a great deal today about risks in airplanes being equal to those in other environments. For example, risk of transmission of infectious disease is no different in an airplane than it is in a subway or an office building. But because risks are equal doesn't make them acceptable if the risks can reasonably be minimized.

Number two: The absence of evidence of health effects associated with specific pollutants measured in aircraft doesn't mean that the effect isn't there. It means that the studies have not been done. There is not one single study that measures or in any way documents health effects along with air quality measurements. All the studies we are hearing about only report measurement of specific pollutants. We haven't heard about any study that correlates measurements of specific pollutants with health effects. Until we have this kind of data, we are not going to know whether or not the air quality in airplanes is actually safe.

Thank you.

Dr. SPENGLER. Let me stress five actions that I think the airlines might do and the manufacturers might do. First of all they can emulate Swiss Air and clean their planes more thoroughly. We were amazed to find dust mite antigen and cat antigen in the aircraft carpet, and seat dust samples. It was very easy to collect the dust from carpets and seats.

Mr. OBERSTAR. Expand on that. What does Swiss Air do and how do they do it?

Dr. SPENGLER. I refer to the passion for cleanliness of the Swiss people. The point being that it is a maintenance practice, as well as an attitude that comes down through the management to say, "We are going to improve the conditions of our planes and the thoroughness with which we will clean our planes." That is my point. It may not be exactly what Swiss Air does per se but I will tell you, Swiss Air has very thorough cleaning procedures and requirements.

We found that without a doubt the highest CO_2 concentrations, and possibly the highest potential for exposure, occurred during the boarding and deboarding process. The boarding process alone can go on for a half-hour to an hour. And obviously it is the flight attendants who are in residence the longest under those conditions.

We feel that the airlines ought to reexamine the ozone catalyst issue. And this will be amplified with the results that Sue Ludwig will discuss. There is now good evidence that the halflife of the catalyst used to remove the ozone from the cabin supply air is fatiguing a lot sooner than what you would expect from the design and maintenance.

There is a clear need to understand the internal mixing patterns of cabin air as a means of transmissions of infectious agents. This

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is not well understood and I think there is a lot of misconception about how planes internally mix.

Finally, I want to say they ought to examine—this is the manufacturers—I think they ought to examine particle filter efficiencies—what it is in practice, not just from design. Also they could explore the use of UV radiation, which is being used very effectively as a control mechanism for killing TB and viruses in hospitals and other health care settings. This has possible application to aircraft. It may result in an extra safety margin, and should be explored.

That is all I have to say.

Mr. OBERSTAR. Thank you very much.

Mr. Halfpenny.

Mr. HALFPENNY. Thank you, Mr. Chairman.

I am sorry I didn't prepare any written presentation. I didn't understand this. I was confused, easily am.

Mr. OBERSTAR. That is quite all right. We don't need written testimony, except for where there is some very technical documentation accompanying it. I always appreciate a witness who comes to this committee and speaks from the fullness of knowledge.

Mr. HALFPENNY. Thank you. I will do my best.

I thought maybe I would start off by reviewing some of the parameters for fresh air ventilation on an airplane. You described them quite well. Air is extracted from the compressor stages of an engine, although the temperatures usually are lower than 1,200, more like 600 or 700. It is then cooled through a precooler and goes through a heat exchanger and an environmental control unit, where it is again cooled down to the proper temperature, say 30 degrees to 150, whatever is required to heat or cool the airplane.

All of these functions are penalties to the airplane. First of all, extracting from the core engine is a fuel burn penalty. It raises the turbine gas temperature about one degree for every 200 pounds. It doesn't sound like much but they are running up near the limit. It is a drag item.

The air that is used to cool it is fan air, and that is a drag item. And the heat exchangers themselves have a drag item effect on the airplane. The motivation, of course, by the manufacturers is to reduce fuel burn, and this is a very sensitive economic issue with airplane builders. A half percent difference in fuel burn makes them competitive or noncompetitive. When you are burning 12- to 14,000 pounds of fuel an hour, a percent over 3,000 hours per year becomes an immense number.

The other thing about this is that after this air goes into the cabin, it is distributed, and Mr. DeFazio brought up a point or one of the gentlemen did about how effective it was. I believe it was the Chairman who asked a question about how effective it is.

This is something that is probably ignored by most designers. ASHRAE deems effectiveness is 100 percent. You say, Well, I am going to put 100 CFM of air into the unit and there is 1 CFM of contaminant per minute, you will have 1 percent. But that is only if it is well mixed. You can have local concentrations.

I did some analysis of some data and talked to Dr. Burge about it. We tend to agree on this, that the effectiveness in cabins ap-

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COSMIC RADIATION DOSES RECEIVED BY AUSTRALIAN COMMERCIAL FLIGHT CREWS AND THE IMPLICATIONS OF ICRP 60

Owen J. Wilson, Beverley F. Young, and Cheryl K. Richardson*

Abstract—In response to concerns expressed in the aviation industry, surveys of the cosmic radiation doses received by Australian commercial flight crews were carried out in 1982–1983 and in 1991. Dose rate measurements were made by use of thermoluminescent dosimeter personal monitors, CR-39 disks and portable monitoring equipment. The results indicated that the average annual doses to domestic crew members were between 1.0 and 1.8 mSv. They further indicated that an increase in annual doses had occurred since 1983. A method of estimating doses from flight records was developed and the results compared favorably with the measurements from both surveys. By use of this method, annual doses up to 3.8 mSv were estimated for international crew members. This represented a significant increase since 1983. It is doubtful that the average doses received by either group would comply with the ICRP Publication 60 public limit of 1 mSv y^{-1} averaged over 5 y. ICRP Publication 60 recommended that the new occupational annual dose limit of 20 mSv should now apply to flight crews. This represented a dose limit of at least four times that which was previously permitted. In view of the general trend to lower dose limits, the nature of the occupation, and given that annual doses were found to be $<$5 mSv, the use of an annual dose limit of 5 mSv is suggested for Australian commercial flight crews.

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Key words: surveys; radiation, atmospheric; dosimetry; International Commission on Radiological Protection

INTRODUCTION

THE COSMIC radiation doses received by the crews of jet aircraft have stimulated considerable interest and debate in the aviation industry, the scientific community, and by the authorities responsible for the regulation of dose limits. Prior to the introduction of Publication 60 of the International Commission on Radiological Protection (ICRP 1991), the dose limits that were generally accepted for flight crews were those appropriate for public exposure. Flight crews were not considered to be radiation workers because they were neither employed nor trained to have any direct involvement with radiation sources. Dose estimates for some crew members who fly for many hours a year at high altitudes and high latitudes could exceed the limit for public exposure. As these doses were received occupationally, the argument was presented that the dose limit for occupational exposure should apply to flight crews (Barish 1990).

Cosmic radiation is natural background radiation and previous criteria excluded this from dose assessments. The doses received by crews represented a non-trivial population dose, considering the large occupational group exposed worldwide. It would have been difficult to ignore these doses on the grounds that they occurred due to exposure to natural background radiation. Reasonable arguments existed for assessing the doses to flight crews by using the limits for either public or occupational exposure and for and against the inclusion of the cosmic doses. In addressing these issues, ICRP recommended that there should be a requirement to include exposures to natural sources as part of occupational exposure only in specific cases, of which the operation of jet aircraft was included, and that, in practice, occupational dose limits are applied to all occupational exposure. ICRP essentially placed flight crews into the occupational exposure group and recommended that the new 20 mSv annual dose limit should now apply.

The recommendations made in ICRP 60 (ICRP 1991) resolved many of the issues but raised others in doing so. Recent increases in the perceived risks from exposure to ionizing radiation resulted in the general reduction of dose limits. Present ICRP estimates indicate a risk of 1 in 20,000 of developing a fatal cancer, per 1 mSv effective dose. The recommended limit for those involved with the operation of jet aircraft, now being at least four times that previously accepted, is in marked contrast to the downward trend. The dose limits applicable to flight crews since 1966 are shown in Table 1. Although exposed at their work place, crew members clearly could not be described as radiation workers. In the absence of an alternative exposure group designation, the assignment of flight crews to the occupational exposure group was necessary because the doses received would have exceeded the new ICRP 60

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Table 1. A chronological review of ICRP recommended dose limits applicable to flight crews indicating that, although public and occupational dose limits have reduced substantially in recent years, the new limit recommended for flight crews is at least four times the previous limits.

Exposure group	Annual limit (mSv)	Recommended publication	Reference
Public	5	ICRP 9	ICRP 1966
Public	5	ICRP 26	ICRP 1977
Public	1[a]	ICRP 45	ICRP 1985
Occupational	20[b]	ICRP 60	ICRP 1991

[a] Lifetime average (e.g., 2 mSv y^{-1} for 35 y, assuming a 70-y lifetime).
[b] The previous ICRP occupational limit (ICRP 1977) was 50 mSv.

public limit of 1 mSv y^{-1}, averaged over 5 y. These issues would not be a problem if doses were independent of limits, but if that were so there would be little point in setting limits. The need to comply with the earlier dose limits put considerable pressure on the aviation industry to minimize doses and to investigate the issues. Practices that could result in unnecessary dose increases may now occur because of the increase in the dose limit and the comparative ease of compliance with that limit.

The interest of the Australian Radiation Laboratory (ARL) in the cosmic radiation doses received by flight crews was first stimulated in 1982 by requests for advice from the aviation industry. The particular areas of concern were the doses received by Australian flight crews, the associated risks, and the appropriate application of recommended dose limits. As there were few measured data in the southern hemisphere, a survey of the doses to Australian international and domestic flight crews was carried out in 1982–1983. The aim of that survey was to test compliance with the dose limits recommended at that time. Changes to the recommended dose limits, deregulation of the industry, and advancements in aircraft design and performance prompted a second survey of domestic crews which was carried out in 1991. The results of these surveys are presented with a view to the following:

- addressing the concerns expressed in the aviation industry;
- evaluating practical methods of assessing and minimizing the doses received by flight crews;
- determining the relativity of the cosmic dose rates in the northern and southern hemispheres;
- investigating the influence of technical advancements in aircraft design, on the doses received; and
- exploring the implications of the new ICRP Publication 60 (ICRP 1991) recommendations.

MATERIALS AND METHODS

Cosmic radiation consists of high-energy charged particles—protons (87%), alpha (11%), heavy nuclei (~1%), and electrons (~1%)—of galactic and solar origin, and their atmosphere interaction products—high-energy muons, electrons, photons, and neutrons (NCRP 1975, 1987; NAS-NRC 1986). Most of the primary radiations are absorbed in the upper atmosphere, leaving the secondary muons, electrons, and photons (often referred to as the ionizing component), and the neutrons.

The cosmic radiation doses received by flight crews vary as a function of altitude, latitude, aircraft construction, solar activity, and exposure period. Most domestic flights within Australia range between 12 and 43 degrees south latitude at altitudes up to 13 km. Australian international flights are between 43 degrees south and 53 degrees north latitude at altitudes up to 14 km. The lightweight construction of an aircraft fuselage provides little shielding from high-energy cosmic radiation but shielding by components such as instrumentation, engines, and fuel may be significant. The wing loading, engine thrust, and fuel economy determine the optimum cruise altitude and speed of an aircraft, which in turn determines the dose rate and exposure period. Diurnal variations in the solar component of cosmic radiation, solar flares, and the solar cycle can add time dependence and uncertainty to cosmic radiation measurements. The measurement strategy was designed to allow for such variables.

Dose rate measurements (1991 domestic survey)

The ionizing dose rates were measured as a function of altitude and latitude across Australia. The measurements were made by use of two Studsvik Gammameter 2414A radiation monitors. These monitors had large scintillator detectors which had appropriate sensitivity and energy response for cosmic radiation measurements. The energy response of this monitor is stated by the manufacturer to be within 10% of the true value from 45 keV and 4 MeV. The monitors were tested and calibrated prior to taking the measurements. The calibration method was traceable to the Australian exposure standard held at ARL.

All measurements were made on scheduled domestic passenger flights. Flights with significant north-south directions were made to determine altitude and latitude effects. Flights with significant east-west directions were made to observe other effects. Concurrent measurements were made in the cockpit and passenger cabin. Altitude and position readings were taken from the altimeter and navigational instruments on the aircraft throughout the flights. The time of every measurement was recorded so that the cabin and cockpit results could be correlated. All of the cockpit measurements were made at a position directly behind the pilot. The cabin measurements were made at one location per flight. The location in the cabin was changed for different flights so that variations in dose rate could be observed. Between flights, the monitor used in the cabin was intercompared and interchanged with the one from

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the cockpit to ensure that observed differences in dose rate were not instrument-related.

Personal monitoring (1991 domestic survey)

One hundred ionizing and 20 ionizing-neutron personal monitors were used in the survey. The measurement period was from 20 November 1990 to 22 May 1991. Each monitor was exposed for 3 mo during the measurement period. Two domestic airlines each carried one-half of the monitors. Measurements were made as a function of flight path, aircraft type, and crew member occupation. The monitors were carried by crew members, except for those used for monitoring aircraft which were installed in the cabin and cockpit of the designated aircraft. The personal monitors were allocated as shown in Table 2. The details of every flight taken during the exposure periods were recorded. Each flight record included the date, aircraft type, departure port, arrival port, departure time, arrival time, period of ascent, period at cruise altitude, period of descent, and the cruise altitude. A total of 6,716 flights were recorded.

The ionizing personal monitors consisted of a plastic holder that contained a $CaSO_4$:Dy thermoluminescent dosimeter (TLD) card. Energy discrimination was provided by four filtered regions of the card. The area of maximum filtration or the high-energy response region of the monitor was used. This provided reasonable dose assessments with minimal sensitivity to the radiation from baggage x-ray units. Monitors that had inadvertently passed through baggage x-ray units were detected by analysis of the filtered regions.

The ionizing-neutron monitors consisted of a plastic holder that contained a 12.7-mm-diameter $CaSO_4$:Dy TLD disk and a CR-39 plastic disk of similar diameter. The TLD disk was used to measure the ionizing component while the CR-39 disk was for the neutrons. The monitors each contained a copper filter for the TLD disk that gave it a uniform energy response and low energy cutoff which minimized the doses from inadvertent exposure in baggage x-ray units. The CR-39 disk was unfiltered, apart from the thickness of the plastic holder. The monitors were calibrated by comparison with the Australian exposure standard held at ARL.

As indicated in Table 2, the 20 ionizing-neutron monitors were installed in the cabin and cockpit of various aircraft. Each ionizing-neutron monitor was accompanied by an ionizing monitor so that a comparison of the results from the two monitors could be made and so that exposure in baggage x-ray units could be detected. The primary functions of the ionizing-neutron monitors were to determine whether or not CR-39 was a viable neutron detector for cosmic radiation measurements on aircraft and, if so, to use them to estimate the relative dose contributions from the ionizing and neutron radiations.

Table 2. Allocation of the ionizing (TLD) and the ionizing/neutron (TLD/CR-39) personal monitors in the 1991 survey.

Subject	TLD	TLD/CR-39
Cockpit crew	30	0
Cabin crew	40	0
Aircraft cockpit	10	10
Aircraft cabin	10	10
Flight path	10	0

Personal monitoring (1982–1983 domestic and international survey)

TLD personal monitors were worn by 47 domestic flight crew members in the 6 mo between 2 January 1982 and 2 July 1982. Both cabin and cockpit crew were monitored. Six international and six domestic crew members were monitored continuously for the 12 mo from 1 May 1982 to 28 April 1983. Each monitor was exposed for 3 mo and then replaced. Flight details were recorded for the 6,600 flights taken by the crew members during the exposure periods.

The monitors used in the survey were of similar design to the ionizing-neutron monitors previously described, except that a second TLD disk had been used instead of the CR-39 disk. The result from this unfiltered TLD was used to indicate inadvertent exposures in baggage x-ray units.

An estimate of the high-altitude neutron component of the dose equivalent was made for the 1982–1983 survey by use of ICRP Publication 26 (1977) and the 1982 report of the United Nations Scientific Committee on the Effects of Atomic Radiation (UNSCEAR 1982). The ICRP recommended the use of a quality factor of 10 when the neutron spectrum was unknown. The average neutron dose equivalent was estimated to be 0.8 of the ionizing dose over the altitude and latitude ranges of interest. Although considered at the time to be a worst-case estimate, subsequent revisions of neutron radiation weighting factors (ICRP 1991) and a better understanding of the cosmic neutron contribution (UNSCEAR 1988) would indicate that 0.8 was a reasonable value to use.

RESULTS AND OBSERVATIONS

Dose rate measurements (1991 domestic survey)

Two flights were made on a Boeing USA 767 which was flown between Melbourne and Darwin. The aircraft landed at Adelaide and Alice Springs en route. The flight path is shown in Fig. 1. The flights were primarily in a north-south direction. The measurements were made in the cockpit and at the front of the cabin for both the outward bound and the return flights. The measured dose rates for the outward bound flight are shown in Fig. 2. During the flight, a cruise altitude of 12.5 km (41,000 ft) was maintained between each port. A reduction in dose rate as a function of latitude can be readily seen as the aircraft traveled towards Darwin.

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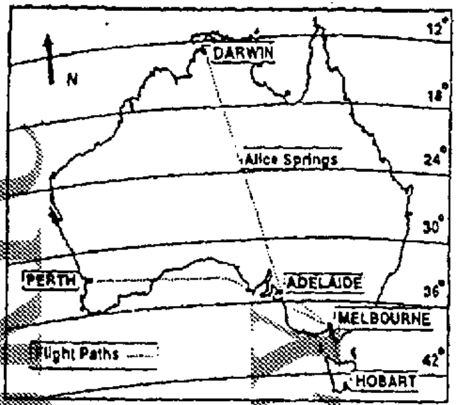

Fig. 1. Map of Australia showing the flight paths and airport locations used for the dose rate measurements.

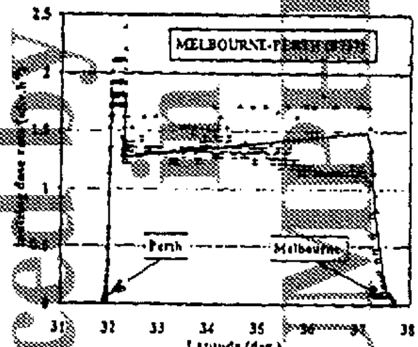

Fig. 2. Ionizing dose rates measured on the flight from Melbourne to Darwin.

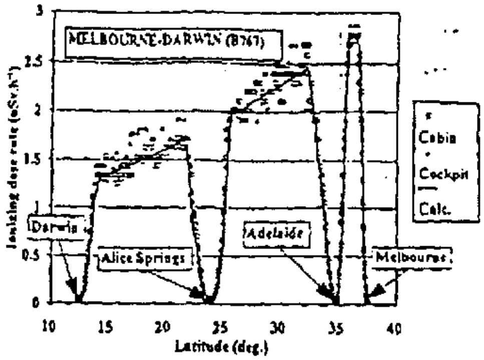

Fig. 3. Ionizing dose rates measured on the flight from Melbourne to Perth.

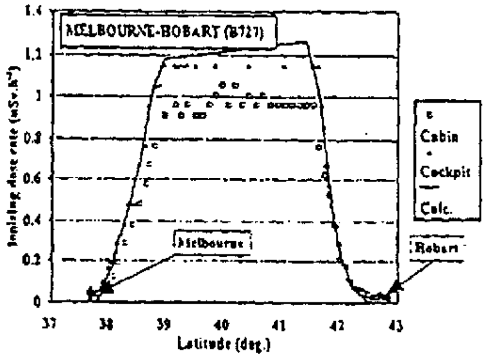

Fig. 4. Ionizing dose rates measured on the flight from Melbourne to Hobart.

The dose rates were ~10% higher in the cockpit than in the cabin.

Two flights were made on a B737 which was flown between Melbourne and Perth. The flight path is shown in Fig. 1. The direction of these flights was primarily east-west. Measurements were made in the cockpit and in the cabin, directly above the wing, for the outward-bound flight. The position in the cabin was moved six rows forward of the wing for the return flight. The measured dose rates for the outward bound flight are shown in Fig. 3. A cruise altitude of 10 km (33,000 ft) was maintained for most of the flight. Approaching the end of the flight, the aircraft was taken to an altitude of 11.3 km (37,000 ft) to allow measurements to be made at the service ceiling of the B737. The corresponding increase in the dose rate can be seen in Fig. 3. The dose rates measured in the cockpit were higher than those in the cabin.

Two flights were made on a B727 that was flown between Melbourne and Hobart. The cabin measurements for these north-south flights were made at the rear of the aircraft. The flight path is shown in Fig. 1. The measured dose rates for the outward-bound flight are shown in Fig. 4. The cruise altitude for this flight was 9.1 km (30,000 ft). Once again the dose rates in the cockpit were higher than those in the cabin.

For most of the flights, a reduction in the measured dose rate was observed as the aircraft left the ground. Dose rates of down to one-half those at ground level were found at altitudes between 250 and 700 m. The effect was also observed while on landing approach. The measurements indicated that at these low altitudes the cosmic dose rate increases slowly with increasing altitude while the relative contribution from the terrestrial radiation source reduces rapidly due to distance and air attenuation. The measured dose rates increased above these altitudes and reached those found at ground level at altitudes ranging from 2.5–3 km above ground level. The ionizing dose rates increased rapidly at altitudes above 3 km, and levels of 1.5 $\mu Sv\ h^{-1}$ and 3 μSv

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...$^{-1}$ were found at 10 km (33,000 ft) and 12.5 km (41,000 ft) above Melbourne, respectively. At the same altitudes above Darwin, the dose rates were found to be less than one-half those above Melbourne.

General aviation in Australia, which includes the use of light aircraft and most aviation other than that of the commercial airlines, is usually carried out below a 3-km ceiling above sea level. The measurements indicated that at these low altitudes the doses received by those participating in general aviation would not vary significantly from the doses received on the ground.

The flights between Melbourne and Perth demonstrated the shielding properties of the wing structure and fuel tanks of the B737. The dose rates measured in the cabin were up to 30% lower than those measured in the cockpit and they appeared to increase as the latitude decreased. At the same time, the dose rates in the cockpit decreased with decreasing latitude, as expected. As the aircraft consumed its fuel supply, the measured dose rate increased and approached that in the cockpit. This observation appears to demonstrate the omnidirectional nature of the scattered cosmic radiation, given that the fuel tanks were below the measurement location and the primary radiation source was above. A similar observation was made on the flights between Melbourne and Hobart. The dose rates found in cabin were ~20% lower than those in the cockpit. It is believed that much of the observed difference was due to the shielding provided by the wings, engines, and their support structure near to the measurement location at the rear of the cabin. Shielding provided by the passengers can also be assumed to contribute to the lower dose rates found in the cabin than in the cockpit.

The solid lines denoted "calc." in Figs. 2, 3, and 4 correspond to the expected dose rate at the front of the cabin of a B767. The method of its determination is described later. From this, it was possible to compare the dose rates found in the B767 with those of the B727 and B737 at the same position and altitude. For each aircraft, the dose rates found in the cockpit were from 10–30% higher than those found in the cabin. The dose rates found in the cabin and cockpit of the B737 were similar to those found in the B767. The dose rates found in the B727 were ~20% lower than those of the B767 and B737. The difference in dose rates may reflect the difference in the materials and design of the B767 and B737 with respect to the older B727.

The measured dose rates were inspected for evidence of diurnal variations. The cockpit measurements from the flights between Melbourne and Perth were considered most likely to demonstrate the effect if it was significant. Measurements made at 1112 local Standard Time, while approaching Perth at an altitude of ~ 7 km (35,000 ft), were compared with those made a... .00 local Standard Time, at the same altitude and latitude, on the return flight from Perth. A difference in the dose rates was not observed. It was concluded that if diurnal effects exist, the variation in the dose rate must be small under normal solar conditions; however, diurnal variations may become evident under solar flare conditions due to the increase in the solar component relative to the galactic contribution.

Dose and dose rate modeling

The measured dose rates from the Melbourne-Darwin flights were used to develop expressions that may be used to calculate doses from flight details. Due to the predominant use of imperial units in the aviation industry and metric units in the scientific community, both units were accommodated in the following expressions.

The dose rate was found to be very nearly proportional to the cube of the altitude, from sea level, up to the range of cruise altitudes of most commercial aircraft. The ionizing effective dose rate, $\dot{E}_{Ref}$ μSv h^{-1}, at altitude, A, up to 12.5 km (41,000 ft) at an arbitrary reference latitude of 36 degrees is

$$\dot{E}_{Ref} = pkA^3, \quad (1)$$

where

$k = 1.41 \times 10^{-3}$ (for A in km); or
$k = 4.0 \times 10^{-14}$ (for A in ft); and
$p = 1$ (cabin); or
$p = 1.1$ (cockpit).

The reference latitude was located between Melbourne and Sydney, which are the ports of the most frequented commercial flight path used in Australia. The correction factor, p, for the difference in the dose rates between the cabin and cockpit positions was included in the expression.

Eqn (1) should not be used for altitudes above 12.5 km because the dose rate begins to level off at these higher altitudes and the expression would overestimate the value. Although the measured cosmic ionizing dose rate at sea level is ~0.02 μSv h^{-1}, it can be seen that the expression predicts zero dose rate at zero altitude. This is because the cosmic dose received by flight crews, while they are on the ground, should not be included in their dose assessment. A value of 0.02 μSv h^{-1} can be added to the estimate from eqn (1) for the purpose of comparison with measurements.

A correction factor, L, for the variation in dose rate as a function of latitude, θ, was obtained by analysis of the measurement results:

$$L = 0.37 + 1.83 \sin^2(\theta). \quad (2)$$

The form of eqn (2) allows the use of positive and negative values of θ to represent latitudes in the southern and northern hemispheres. This can be useful when considering flights that cross the equator.

The ionizing effective dose rate, $\dot{E}_\gamma$, at the altitude and latitude of interest, can be calculated using

$$\dot{E}_\gamma = \dot{E}_{Ref}L. \quad (3)$$

The solid lines shown with the measured data points in

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Figs. 2, 3, and 4 were calculated by use of eqn (3), from the flight navigation readings taken at the time of each measurement. The cosmic ionizing dose rates, which Australian domestic flight crews can expect to encounter as a function of altitude and latitude, were calculated by use of eqn (3) and are shown in Fig. 5.

Dose rate measurements made between 35 and 50 degrees north latitude, at a cruise altitude of 12.5 km (41,000 ft) (Hewitt et al. 1978) were recorded at the same cruise altitude as the Melbourne-Darwin flights, which were between 12 and 38 degrees south latitude. It was found that the measurements made in the northern hemisphere matched those in the southern hemisphere where the corresponding latitudes overlapped. The values obtained by use of eqn (3) were consistent with the measurements over the full 12-50 degrees latitude range. The results indicated that the cosmic ionizing dose rates in the northern and southern hemispheres were very similar. Assuming that dose rates do not deviate significantly from those estimated between the equator and 12 degrees latitude, the expression can be expected to give reasonable estimates of the ionizing dose rates between 50 degrees south and 50 degrees north latitudes. The latitude range of eqn (3) covers all Australian domestic and international flights.

The integral of eqn (3) with respect to time,

$$E_\gamma = \int \dot{E}_{Ref} L\, dt \qquad (4)$$

may be used to estimate the accumulated dose expected for a given flight. Eqn (4) may be simplified, as shown:

$$E_\gamma = L_{Ave} \int \dot{E}_{Ref}\, dt, \qquad (5)$$

replacing L with its average value, L_{Ave}, which is given by

$$L_{Ave} = \frac{\int L\, d\theta}{(\theta_2 - \theta_1)}, \qquad (6)$$

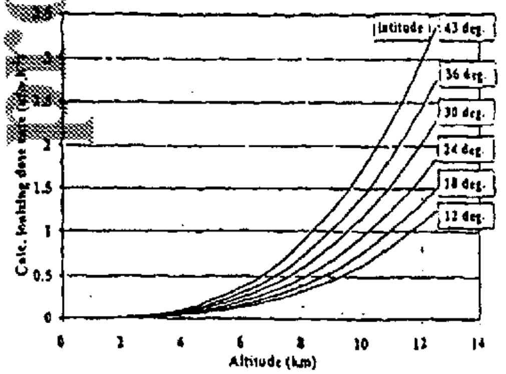

Fig. 5. Ionizing dose rates in Australia that were estimated as a function of altitude and latitude, by use of eqn (3).

where

θ_1 = latitude (radian) of the departure port; and
θ_2 = latitude (radian) of the arrival port.

For short flights, the variation in L is small. For flights that are sufficiently long for the variation in L to become significant, most of the dose is received at cruise altitude where $\dot{E}_{Ref}$ is constant. In each case the error introduced due to the approximation is small. By substituting eqn (2) into eqn (6) and evaluating the integral, the average latitude correction becomes

$$L_{Ave} = 1.285 + \frac{0.915\,[\sin(\theta_1)\cos(\theta_1) - \sin(\theta_2)\cos(\theta_2)]}{(\theta_2 - \theta_1)}. \qquad (7)$$

In order to evaluate eqn (5), expressions for the variation of altitude as a function of time are required. An aircraft on a regular domestic flight follows a semi-parabolic ascent path, cruises at a constant altitude, and descends at a constant rate, as described by eqns (8), (9), and (10), respectively.

Ascent: $(0 < t < T_0)$

$$A = \frac{(bT_0 - A_c)(t - T_0)^2}{T_0^2} + b(t - T_0) + A_c, \qquad (8)$$

where

A_c = cruise altitude;
b = 0.152 (for A and A_c in km), or 500 (for A and A_c in ft);
t = time (min); and
T_0 = ascent duration (min).

Cruise: $(0 < t < T_1)$

$$A = A_c, \qquad (9)$$

where T_1 = cruise duration (min).

Descent: $(0 < t < T_2)$

$$A = A_c \frac{(T_2 - t)}{T_2}, \qquad (10)$$

where T_2 = descent duration (min).

Eqns (8), (9), and (10) were substituted into eqn (5) and the integral was evaluated, giving the ionizing effective dose in µSv:

$$E_\gamma = \frac{L_{Ave} kp(d_0 + d_1 + d_2)}{60}, \qquad (11)$$

where

$$d_0 = \frac{T_0(64\,A_c^3 - 38\,A_c^2\,bT_0 + 10\,A_c b^2 T_0^2 - b^3 T_0^3)}{140},$$

$d_1 = A_c^3 T_1$; and

$$d_2 = \frac{A_c^3 T_2}{4}.$$

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A factor of 1.8 was used to obtain a conservative estimate of the total effective dose, including neutron contribution:

$$E_{\gamma,n} = 1.8\, E_{\gamma}. \quad (12)$$

International flights may include steps in the cruise altitude, which are determined by parameters such as fuel load, gross weight, and air traffic control requirements. Most Australian domestic flights follow direct paths, with minor course corrections en route. This assumption cannot be made for long-haul international flights. Great circle flight paths, which appear on the map to curve toward the nearest pole, may be used to minimize flight distances. When compared with a straight-line path on the map, the curved flight paths should increase the average dose rate marginally but reduce the exposure period. For the purpose of calculating the dose received during long, stepped and/or curved flights, any flight path may be represented by a number of shorter straight paths. Eqn (11) was therefore presented in a modular form so that path corrections could be accounted for with ease.

Personal monitoring measurements:

The measured doses from the 1982–1983 survey are presented in Fig. 6. Those from the 1991 survey are in Fig. 7 for the flight attendants, engineers, and pilots, in Fig. 8 for the aircraft. Difficulties encountered with the flight path measurements of the 1991 survey rendered their outcome unreliable and these data were excluded. The measured doses are plotted with respect to those which were calculated, by use of eqn (12), from the flight data recorded for each monitor. The line of expectation plotted in each figure is that where a measured dose would be plotted if coincident with the calculated value. Averages of the measured and calculated doses are presented in Table 3.

It can be seen from Figs. 6–8 that although the individual measurements could vary significantly from the expected values, their distributions about the lines of expectation were reasonably uniform. This was also indicated by Table 3, where the averages of the measurements were all within 10% of those estimated by use of eqn (12). Conventional personal monitoring would not appear to be a highly accurate method of determining the cosmic dose received by individual crew members. The average dose derived from a large number of personal monitors, however, appears to be reliable. The method of estimating cosmic doses from flight records, as previously described, is not subject to the uncertainties involved with personal monitoring. Dose estimates for individuals can be assumed to be of similar reliability as the average dose estimates.

The annual doses to flight attendants and pilots appear to have increased since the 1982–1983 survey but those for the flight engineers have not changed appreciably. As the measurements from both surveys were consistent with estimates based on measurements made in the 1991 survey, it seems unlikely that the

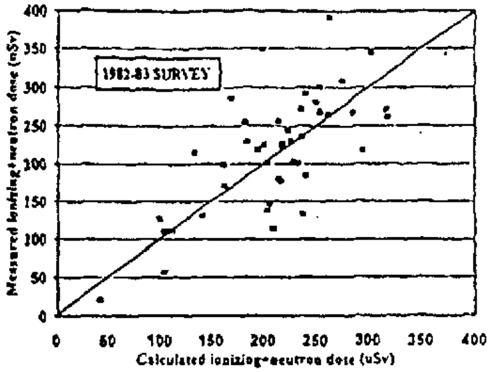

Fig. 6. Ionizing + neutron doses from the 1982–1983 survey compared with doses estimated from flight records, by use of eqn (12).

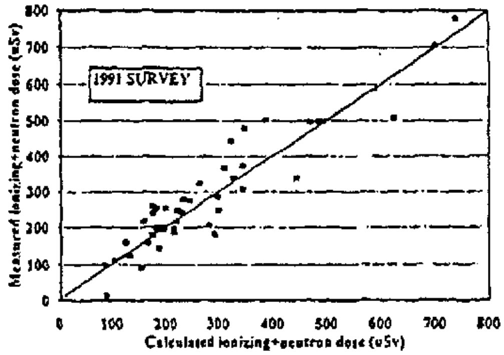

Fig. 7. Ionizing + neutron doses received by flight attendants, flight engineers, and pilots during the 1991 survey compared with doses estimated from flight records, by use of eqn (12).

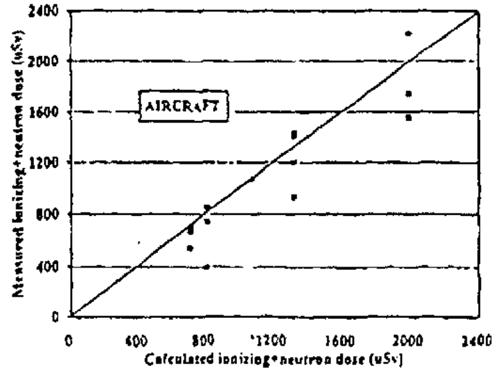

Fig. 8. Ionizing + neutron doses found on aircraft, during the 1991 survey, compared with doses estimated from flight records, by use of eqn (12).

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Table 3. Measured and calculated average dose rates (ionizing and neutrons) from the 1982–1983 and 1991 surveys.

Group	Date	Measured ($\mu Sv\ d^{-1}$)	Calculated ($\mu Sv\ d^{-1}$)	Measured ($mSv\ y^{-1}$)	Calculated ($mSv\ y^{-1}$)
All	1982–1983	2.7	2.5	1.0	0.9
Attendants	1991	4.0	3.9	1.5	1.4
Engineers	1991	3.0	2.7	1.1	1.0
Pilots	1991	4.8	5.0	1.8	1.8

increases were due to variations in the solar cycle. The results can be viewed in terms of the advancements in aircraft technology. The dose rate in a B727 was found to be lower than in the more modern, higher-flying B737 or B767. Flight engineers are more often found in the older aircraft. Flight attendants and pilots are of course found in all of these aircraft. It can be assumed that as the older aircraft are phased out, doses will increase accordingly.

If personal monitoring of flight crews becomes a requirement, as is the case for many in the occupational exposure group, there are a number of important considerations involved. Dose limits refer to doses received in excess of natural background. The assessment of background levels can introduce considerable difficulties in the case of flight crews. The inadvertent exposure of monitors to other radiation sources, such as baggage x-ray units, and the low sensitivity of neutron personal monitors are further considerations.

Control monitors are used to measure background levels which are subtracted from the personal monitor results. In most working environments these can be located near the personal monitors when not in use. For flight crews, the personal and control monitors may be separated by thousands of kilometers. Normal background at ground level is not a constant and, depending on the location, ionizing dose rates from 0.05–0.2 $\mu Sv\ h^{-1}$ or from 1–5 $\mu Sv\ d^{-1}$ can be readily found. Average cosmic ionizing dose rates for domestic flight crews were found to range from 1.4–2.8 $\mu Sv\ d^{-1}$. Although the wearer may travel to various locations and averaging of background levels will occur, significant uncertainties in individual results can be expected due to variations in the large background component of the measured dose. The distributions of the measured doses about the lines of expectation, seen in Figs. 6–8, are believed to be caused primarily by background variations.

The exposure of personal monitors in baggage x-ray units is a major source of uncertainty. The prevalence of this security device at airports presents a major risk that monitors will pass through. The exposure may not be realized by the crew member, due to the routine nature of security procedures involving the units. Documentation of the event in the flight records cannot be relied upon. The dose received by a monitor in passing through an x-ray unit depends on the particular machine and its operational settings. An accurate method of correcting for these exposures could not be found. Monitors suspected of having passed through x-ray units were excluded from the survey. A reliable method of preventing the exposure of monitors in these x-ray units would be necessary if routine personal monitoring is considered for flight crews.

From an analysis of the energy-dependent regions of the 1991 personal monitors, it was suspected that many of those used by the cabin crew may have inadvertently passed through baggage x-ray units, while only a small proportion of those used by the cockpit crew appeared to have been affected. Cabin crew have more direct contact with passengers than do the cockpit crew. In order to comply with airline dress code and to avoid unnecessary passenger concerns, many cabin crew members carried their monitors in hand luggage or in jackets, which could be placed in on-board lockers, instead of wearing the monitor. Monitors carried onto the aircraft were at much higher risk of exposure in baggage x-ray units than those worn by crew members.

Of the 20 ionizing-neutron monitors installed in the aircraft, only two of them recorded neutron doses and those were each 170 μSv. The corresponding ionizing doses were 780 μSv and 620 μSv. The range of ionizing doses measured by the 20 monitors was from 300–970 μSv. The lower limit of detection of the CR-39 disks was estimated to be 80 μSv. It was assumed that their sensitive range covered from one-half to one-third of the cosmic neutron energy range. The lower limit of detection therefore corresponded to a cosmic neutron dose of up to 240 μSv. The measured neutron doses of 170 μSv corresponded to cosmic neutron doses of up to 510 μSv, which was reasonably consistent with the ionizing-neutron dose ratio assumed in eqn (12). An unknown cosmic neutron dose of $<240\ \mu Sv$ can be estimated for those monitors that did not register a neutron dose. These results indicate that the estimated ionizing-neutron ratio of 1.8 was a reasonably conservative value to use. Considerable developmental work seems necessary if CR-39 is to become a useful personal monitor for flight crews.

DISCUSSION AND CONCLUSIONS

It is recognized that there is no dose limit below which absolute safety can be guaranteed. The recommended dose limits are those below which the benefits should outweigh the risks. Doses should be kept below the limits and as low as reasonably achievable, economic and social factors considered. Based on current

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risk estimates, the survey results indicate that an average domestic flight crew member runs a risk of <1 in 10,000 y^{-1} of developing a fatal cancer due to occupational exposure. From dose estimates obtained by use of eqn (12), a person would need to fly for ~900 h at an altitude of 12.5 km (41,000 ft) above Melbourne to receive a dose of 4.9 mSv. For Australian international flight crews, this dose is estimated to be equivalent to that received from 33 round-trip flights between Melbourne and London, which would involve >1,300 h flying time. An upper estimate of the annual doses received by Australian international crew members was made by assuming 1,000 h flying time per year. This gives a dose of 3.8 mSv and a risk of 1 in 5,300 y^{-1} of exposure. The survey results demonstrated that neither the domestic nor the international crew members are likely to exceed annual doses of 5 mSv, for which the estimated risk is 1 in 4,000.

The average annual doses from Table 3 are comparable with average doses received by Australian radiation workers (Morris 1993), such as uranium miners (1.8 mSv) and industrial radiographers (1.7 mSv), and are higher than the doses received by most people. As flight crews are clearly not radiation workers, it would seem difficult to justify further increases of their annual doses, in terms of risk and benefit, considering the social and economic factors involved; however, further dose increases are on the horizon. The indications are that advancements in aircraft technology have increased the doses to flight crews since the 1982–1983 survey, which may be pointing to long term dose increases if future aircraft fly higher and for longer distances. Substantial increases could occur if commercial supersonic aircraft become commonplace. Proposals of hypersonic aircraft, which would fly orbital trajectories above the earth's atmosphere, add to the notion of higher flying aircraft and higher dose rates. When such advancements occur, it is reasonable to expect that the annual doses to flight crews would increase significantly if the recommended limit allows room for such increases to occur. It is necessary that an appropriate dose limitation strategy be in place when this technology arrives so that we are in a position to ensure that it is used with minimal risk.

If we investigate the implications of the recommendations made in ICRP Publication 60 (ICRP 1991) in view of the information just discussed, there appears to be a number of areas of concern. The recommendations now place flight attendants, flight engineers, and pilots in the same exposure or risk group as employees of the radiation industry (such as uranium miners, nuclear power workers, and industrial radiographers). Scope for substantial dose increases has been introduced. The recommendations made for flight crews could be cited as a precedent for other occupations. It could be argued that instead of introducing appropriate measures to comply with the new public exposure limit, the much easier and cheaper option to reclassify the exposed individuals now exists. The situation of flight crews is partly compatible with both of the exposure group definitions but is not fully compatible with either. It would seem that only a partial solution has been achieved by changing flight crews from the public exposure group to the occupational exposure group.

There are a number of regulatory mechanisms by which the perceived difficulties could be overcome by either local authorities or the aviation industry. In order that doses to flight crews are kept to a minimum, in the long term, a limit that does not allow room for substantial dose increases is a necessary component of the solution. A general solution would be to divide the present occupational exposure group into subgroups, such as "radiation worker" and "nonradiation worker." The annual dose limit for the former subgroup would remain at 20 mSv while a previous recommendation for public exposure, at an annual limit of 5 mSv, is suggested for the latter. Australian domestic and international flight crews would appear to have little difficulty in complying with a 5 mSv annual limit. This would allow a modest margin for fluctuations in dose rate due to solar flares and the solar cycle but would ensure that substantial increases could not occur in the future.

Acknowledgements—The authors wish to express their appreciation to the management and staff of Ansett and Australian Airlines; the associations representing flight attendants, flight engineers, and pilots; and to the members of the ARL Personal Radiation Monitoring Service (PRMS). In particular, the assistance of Rhonda Rose of the Australia Flight Attendants Association, Paul Hurst (Ansett Airlines), Robert Sweeting (Australian Airlines), and Neil Morris (ARL PRMS) is acknowledged.

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U.S. Department of Transportation

REPORT TO CONGRESS AIRLINE CABIN AIR QUALITY

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Washington, D.C. 20590

February 1987

Report of the U.S. Department of Transportation to the United States Congress pursuant to Public Law 98-466

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US Department of Transportation
Federal Aviation Administration

Office of the Administrator

800 Independence Ave S W
Washington D C 20591

FEB 27 1987

The Honorable George Bush
President of the Senate
Washington, DC 20510

Dear Mr. President:

I am pleased to submit "The Airliner Cabin Environment--Air Quality and Safety" report prepared in response to Public Law 98-466, dated October 11, 1984. The Department of Transportation (DOT) was directed to commission the National Academy of Sciences (NAS) to conduct an independent study on the cabin air quality in airliners and to submit the study to Congress, along with any comments and recommendations for legislative, regulatory, or industry changes.

The NAS study, which focuses on all health and safety aspects of airline cabin air quality, covers five general subjects: cabin air quality, cabin environment, emergency procedures, regulations, and records. It summarizes the findings of the NAS Committee on Airliner Cabin Air Quality and outlines 21 recommendations for consideration by DOT.

The Department has now completed its review of the NAS recommendations. An Executive Summary and a Summary of Recommendations and Conclusions are contained in the enclosed "Airline Cabin Air Quality" report.

I have sent an identical letter to the Speaker of the House of Representatives.

Sincerely,

Donald D. Engen

Donald D. Engen
Administrator

2 Enclosures

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US Department of Transportation

Federal Aviation Administration

Office of the Administrator

800 Independence Ave. S.W.
Washington, D.C. 20591

FEB 27 1987

Dr. Frank Press
President
National Academy of Sciences
2101 Constitution Avenue, NW.
Washington, DC 20418

Dear Dr. Press:

The Department of Transportation (DOT) has completed its review of "The Airliner Cabin Environment--Air Quality and Safety" report prepared by the National Academy of Sciences (NAS) in response to Public Law 98-466.

The enclosed report, "Airline Cabin Air Quality," provides DOT's response to the 21 recommendations of the Committee on Airliner Cabin Air Quality (the Committee). DOT has accepted in full or in part most of the recommendations of the Committee.

Although research is still needed in the area of cabin air quality, the NAS study has laid the necessary groundwork. Please convey my appreciation to the members of the Committee.

I have also sent copies of both reports to the Speaker of the House of Representatives and the President of the Senate.

Sincerely,

Donald D. Engen

Donald D. Engen
Administrator

Enclosure

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EXECUTIVE SUMMARY

Public Law (P.L.) 98-466, dated October 11, 1984, directed the Secretary of Transportation to commission an independent study by the National Academy of Sciences (NAS) on the cabin air quality in airliners and to submit a copy of that study, together with any comments and recommendations to Congress.

In compliance with P.L. 98-466, a contract was negotiated between the Federal Aviation Administration (FAA) and the NAS. The NAS formed the Committee on Airliner Cabin Air Quality (the Committee) to study all safety aspects of airline cabin air quality and submitted its report, "The Airliner Cabin Environment--Air Quality and Safety," to the FAA on August 12, 1986. The NAS study focuses on all health and safety aspects of airline cabin air quality. The report includes recommendations for legislative, regulatory, and industry changes in relation to airline cabin air quality. The Executive Summary of the NAS report contains the Committee's findings and recommendations.

This report provides the Department of Transportation's (DOT) response to the 21 recommendations made by the Committee. We have accepted in full or in part most of the recommendations of the Committee. Three major issues of special interest--a proposed ban on smoking, further study of cabin air quality, and Federal responsibility for health effects associated with air travel--are addressed below:

1. Proposed Ban on Smoking

   The Committee recommended a ban on smoking on all commercial domestic flights.

   While DOT recognizes that exposure to environmental tobacco smoke (ETS) could be viewed as a problem by some crew and passengers, we believe that further study is needed before the Department can propose a definitive response to this recommendation.

   Issues that require further consideration include the following:

   - Further review of the health effects of ETS, including review of the studies and reports completed since the NAS study.
   - Further review of the concentration and distribution of pollutants for various aircraft types.
   - Additional consideration of possible technological solutions (which the Committee found infeasible) to determine if modifications to aircraft ventilation systems or procedures would provide acceptable results.
   - Further consideration of possible application of any proposed solutions to international, as well as domestic, flights.

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2. Study of Cabin Air Quality

A major finding of the Committee was "...that, if the lowest rate of ventilation permitted by current equipment design were used under conditions of full or nearly full passenger loads, the resulting ventilation rate would be at the minimum determined to provide acceptable air quality when smoking is not permitted and other contaminant sources are not present. In the absence of sources of contamination, this rate does not constitute a health hazard."

Based on this finding, the Committee recommended that "A data collection program that measures airflow and contamination in airplanes should be implemented."

DOT concurs with the Committee's finding. Discussions, which began in May 1984, will be renewed with the Environmental Protection Agency. A cooperative effort will be made to determine the feasibility of initiating a practical program for making cabin air quality measurements on board airplanes during typical operations. Contaminants from all sources will be considered as a major item in the conception of this program.

3. Federal Responsibility for Air Travel Health Effects

The finding of the Committee that no Federal agency has direct responsibility for dealing with health effects associated with air travel was based on misinformation. In fact, the Department does have authority through FAA and the Office of the Secretary covering both crew and passenger health. The FAA has the authority to regulate the health aspects of an aircraft in operation. In addition, the Office of the Secretary is required by law to ensure that airlines provide "safe and adequate service." Other Federal agencies have complementary health responsibilities. The responsibilities of these agencies do not conflict with those of DOT.

Given the broad range of DOT authority over passenger and crew health, as well as the expertise and the nature of the missions of the other agencies which have health responsibilities, we see no reason to consolidate or transfer any authority. We recommend retention of the present system of health responsibilities.

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SUMMARY OF RECOMMENDATIONS/CONCLUSIONS

The following is a summary of the recommendations made by the Committee followed by DOT's conclusions/recommendations to Congress (in bold type). Section II of this report contains a detailed commentary of the reasons for DOT's recommendations and conclusions. For reference purposes, the page number corresponding to each recommendation contained in Section II is included at the end of each DOT recommendation.

1. "The Committee believes that the health effects associated with air travel should be within the purview of a Federal agency."

   The finding of the Committee that no Federal agency has direct responsibility for dealing with health effects associated with air travel was based on misinformation. In fact, the Department does have authority through FAA and the Office of the Secretary covering both crew and passenger health. Other Federal agencies have complementary health responsibilities. The responsibilities of these agencies do not conflict with those of DOT.

   Given the broad range of DOT authority over passenger and crew health, as well as the expertise and the nature of the missions of the other agencies which have health responsibilities, we see no reason to consolidate or transfer any authority. We recommend retention of the present system of health responsibilities.

   (Reference Page 3)

2. "The Committee found that, if the lowest rate of ventilation permitted by current equipment design were used under conditions of full or nearly full passenger loads, the resulting ventilation rate would be at the minimum determined to provide acceptable air quality when smoking is not permitted and other contaminant sources are not present. In the absence of sources of contamination, this rate does not constitute a health hazard."

   This was a finding of the Committee and requires no action.

   (Reference Page 5)

3. "A data collection program that measures airflow and contamination in airplane cabins should be implemented."

   Discussions, which began in May 1984, will be renewed with the Environmental Protection Agency. A cooperative effort will be made to determine the feasibility of initiating a practical program for making cabin air

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quality measurements on board airplanes during typical operations. Contaminants from all sources will be considered as a major item in the conception of this program. Another topic that will be included in these discussions is whether the many variables of flight can be monitored or controlled to correlate test results from different flight conditions.

(Reference Page 6)

4. "The FAA standard (on carbon dioxide) is much higher than standards for other confined environments. The Committee recommends that the FAA review its carbon dioxide standard."

The FAA will review the need to establish either ventilation limits, reduced carbon dioxide limits, or both. After the review, any needed rule change and advisory material will be proposed.

(Reference Page 7)

5. "Therefore, the Committee suggests that FAA carry out a carefully designed program to ensure that cabin ozone concentrations comply with Department of Transportation regulations."

The FAA will issue biennial action notices requiring FAA inspectors to report on the present status of all U.S. air carriers' compliance with the existing ozone regulations. The responses to the action notices will be summarized and published. Identified deficiencies will be corrected.

(Reference Page 8)

6. "The Committee recommends a ban on smoking on all domestic commercial flights, for four major reasons: to lessen irritation and discomfort to passengers and crew, to reduce potential health hazards to cabin crew associated with ETS (environmental tobacco smoke), to eliminate the possibility of fires caused by cigarettes, and to bring the cabin air quality into line with established standards for other closed environments."

The Committee stated that "Empirical evidence is lacking in quality and quantity for a scientific evaluation of the quality of airliner cabin air or of the probable health effects of short or long exposure to it."

We agree that exposure to ETS could be viewed as a problem by some crew and passengers. However, we believe that further study is needed before the Department can propose a definitive response to this recommendation.

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Issues that require further consideration include the following:

- Further review of the health effects of ETS, including review of the studies and reports completed since the NAS study.

- Further review of the concentration and distribution of pollutants for various aircraft types.

- The effectiveness and economic effects of possible technological solutions such as modifications to aircraft ventilation systems or procedures.

- Further consideration of possible application of any proposed solutions to international, as well as domestic flights.

(Reference Page 10)

7. "Because a likelihood of occurrence of epidemic disease when forced-air ventilation is not available on the ground has been demonstrated, the Committee recommends that a regulation be established that requires removal of passengers from an airplane within 30 minutes or less after a ventilation failure or shutdown on the ground and maintenance of full ventilation whenever onboard or ground air-conditioning is available."

Because the occurrence of complete ventilation cessation on passenger-laden airplanes is extremely rare and sometimes unavoidable, we do not believe that regulatory action is necessary. However, there may be value in bringing this concern to the attention of the air carriers. The FAA will advise air carriers of the need to deplane passengers, if possible, after 30 minutes without ventilation.

(Reference Page 12)

8. "The Committee also recommends that maximal airflow be used with full passenger complements to decrease the potential for microbial exposure and that recirculated air be filtered (to remove particles larger than 2-3 um) to reduce microbial aerosol concentrations."

The program for making cabin air quality measurements identified under Recommendation 19 will include microbial aerosol concentrations at different cabin airflows. Whether or not maximal airflow is required at all times will be evaluated at the conclusion of this measurement program.

(Reference Page 13)

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9. "The Committee concluded that current pressurization criteria and regulations are generally adequate to protect the traveling public. However, the medical profession should use a more efficient system to warn those with existing medical conditions who are more susceptible to changes in pressure or to long exposure to low pressure that there might be some hazard to their health."

The FAA will continue its ongoing effort to provide aviation medical information to the members of the medical profession to enhance their ability to advise those patients at risk concerning possible hazards secondary to the in-flight environment. In addition to direct contact with FAA-designated aviation medical examiners, the FAA will continue to encourage publication of such information in medical journals. The American Medical Association will be asked to republish the excellent article "Medical Aspects of Transportation Aboard Commercial Aircraft" (Journal of the American Medical Association, February 19, 1982, Volume 247, Number 7) (Appendix 3).

(Reference Page 14)

10. "FAA should consider rule-making that restricts exposure [to cosmic radiation] of pregnant flight crew and crew members. In addition, FAA should investigate total radiation exposure of flight crew and cabin crewmembers through the use of a statistical sample of full-time employees and should require airlines to provide precautionary information to their flight attendants about radiation exposure."

The FAA is currently developing advisory information to promote radiation safety through educational initiatives for use by the air carrier industry. This information will be distributed, and with management cooperation, crewmembers concerned with radiation exposure will be able to limit such by scheduling within domestic and flag route systems.

(Reference Page 15)

11. "The Committee approves of current efforts to base passenger safety briefings and written materials on empirical testing of comprehension and retention."

This recommendation does not require DOT action.

(Reference Page 16)

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12. "The Committee suggests that FAA or appropriate industry organizations consider the advisability of developing an empirical research program to examine passenger response to safety instructions under routine and emergency conditions and revise them as appropriate. Consideration should be given to running some quizzes during a flight to see, for example, what proportion of passengers have retained the key features of the safety briefing."

Attentive passengers who have received the preflight briefings could "pass" quizzes given during nonstressful flight situations, however, that does not necessarily mean that they will take the correct action in a given emergency situation.

The FAA is deeply interested in suggested improvements for briefing techniques from the the airline industry but believes that requiring a quiz data base is not appropriate.

The guidance concerning these briefings is contained in AC 121-24. This AC will be updated as necessary.

(Reference Page 16)

13. "The Committee recommends that FAA require that information on proper response to fire emergencies be included in oral and written passenger safety information."

Recent occurrences of unwarranted passenger-initiated emergency evacuations have caused concern that preflight briefings may in some cases motivate some people to act independently and unnecessarily. The accepted practice of both the FAA and the airline industry is that briefings concerning emergency actions should not create passenger apprehension or inspire unwarranted actions by passengers. The emergency procedures for passengers are, therefore, relatively passive (i.e., follow instructions given for their individual protection) while trained crewmembers implement procedures and deploy any required equipment necessary to assure the continued safe flight and landing of the airplane and the safety of the passengers. The FAA will continue the present practice.

(Reference Page 17)

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14. "The Committee feels that continuing research (fire safety) is also needed in materials development."

The FAA agrees that continuing research is needed in the development of improved fire-safe materials. The FAA's part in this research generally entails the analysis of fire-safety problems and the development and validation of practical small-scale test equipment that can be used to measure reliably the relative fire-safety characteristics of materials. Using this test equipment, the FAA cooperates with material producers, interior fabricators, aircraft manufacturers, and airlines in the development and adaptation of new materials which have a high level of fire safety as well as durability, cleanability, appearance, and other practical characteristics necessary for economically competitive materials.

Recent examples of this highly-successful, cooperative approach between FAA and industry include the establishment of new standards for heat-resistant evacuation slide materials, cargo compartment fire barrier liner materials, fire-safe seat cushion materials, and cabin wall and ceiling panel materials.

The FAA plans to continue this approach in the development of improved materials and believes that this action is responsive to the Committee's recommendation.

(Reference Page 18)

15. "The Committee noted that current emergency procedures for smoke removal recommend that the cabin be depressurized to 10,000 ft. This procedure is ineffective and should be discontinued."

The FAA will obtain and review additional information that is known to address the smoke evacuation issue. This will include a Lockheed study that investigated decompression as a means to control or extinguish a fire, pertinent information retained by the cognizant aircraft certification offices, and any additional information from the NAS. NTSB in-flight smoke and fire accident/incident reports will also be reviewed. Based on this review, the FAA will determine the effectiveness of the current emergency procedures for smoke removal.

AC 25-9 will be revised to be more specific relative to decompression procedures and include any limitations on their use.

(Reference Page 19)

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16. "However, there are generally more crew members than fire extinguishers, and the Committee recommends that FAA review the proposed rule on protective breathing devices for crew members to ascertain the desirability of supplying such equipment for all crew members, rather than limiting it to the persons expected to be involved in firefighting. In addition, the Committee suggests further evaluation of the potential of emergency breathing equipment for all cabin crew members to improve safe and expeditious evacuation of passengers in fire emergencies."

The evaluation of the potential of emergency breathing equipment for all cabin crewmembers to improve the safe and expeditious evacuation of passengers in fire emergencies will be conducted in conjunction with the planned action on Recommendation Number 17.

The FAA is currently participating in a joint international effort between representatives of the Civil Airworthiness Authorities of Canada, France, and the United Kingdom. A meeting will be held in May 1987 between the four nations to decide on further activity. Once this international effort is completed, the FAA will assess the need for regulatory action.

(Reference Page 20)

17. "The Committee recommends that FAA re-examine passenger protective breathing devices and consider requiring that such equipment be available in case of in-flight and postcrash fires."

A joint international effort between representatives of the Civil Airworthiness Authorities of the United States (FAA), Canada, France, and the United Kingdom on passenger protective breathing equipment will continue.

Several meetings have been held during the last 6 months. The groups' objective is to perform a worldwide accident and incident analysis to fully summarize fire events and human factors, in order to evaluate the safety potential of individual passenger protective breathing devices under different fire scenarios. The group will also begin development procedures for testing the devices.

A meeting will be held in May 1987 between the four nations to decide on further activity. Once this international effort is completed, the FAA will assess the need for regulatory action.

(Reference Page 21)

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This information will be used to determine future need for the carriage of emergency medical equipment on air carrier aircraft. This data will also be available for use in studies comparing medical incidents on board air carrier aircraft with those in other settings. The FAA will not undertake these comparative studies since it does not have the authority. Such studies would be inappropriate for the FAA.

(Reference Page 26)

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SECTION II.

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NATIONAL ACADEMY OF SCIENCES' RECOMMENDATIONS
DOT COMMENTS AND PLANNED ACTIONS

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SECTION I.

INTRODUCTION

This report provides the Department of Transportation's (DOT) response to the recommendations made by the National Academy of Sciences (NAS) in its report, "The Airliner Cabin Environment--Air Quality and Safety," dated August 13, 1986.

Public Law (P.L.) 98-466, dated October 11, 1984, directed the Secretary of of Transportation to commission an independent study by the NAS on the cabin air quality in airliners and to submit a copy of that study, with any comments and recommendations to Congress.

Since this was to be an independent study, the Federal Aviation Administration (FAA) did not participate or take any action that could affect the findings, conclusions, or recommendations of the study. At the request of the NAS, however, the FAA provided data and assistance during the course of the study.

Each of the NAS recommendations has been assigned a number to correspond with the sequence in which they are enumerated in the Executive Summary of the NAS report. The DOT report is formatted so that each NAS recommendation is quoted, followed by subsections labeled Comments and Planned Action.

A chronology of the events leading to this report follows:

- May 1980 — Xenex Corporation (Hawaii) petitioned to add requirements for a specified amount of fresh air in cabin, humidification, and air contamination.
- May 1981 — Petition denied.
- October 1981 — S.1770 introduced by Senator Inouye (Hawaii) called for study on same issues as Xenex petition, as well as four other areas of consideration: emergency breathing equipment, removal of smoke and toxic fumes, safe pressurization limits, and collection of medical statistics.
- May 1982 — Hearing before Senate Committee on Commerce, Science, and Transportation, Subcommittee on Aviation.
- January 1983 — S.197 introduced by Senator Inouye identical to S.1770.
- February 1983 — H.R.1333 introduced by Congressman Heftel (Hawaii) identical to S.1770.

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o November 1983 — Hearing before Senate Committee on Commerce, Science, and Transportation, Subcommittee on Aviation on S.197.

o May 1984 — FAA requested EPA assistance to study cabin air quality (later withdrawn due to enactment of P.L. 98-466).

o October 11, 1984 — P.L. 98-466 enacted--Three more areas of consideration added: Exposure to radiation, adequacy of safety instructions, and comparison with foreign industry.

o January 29, 1985 — FAA/NAS contract (DTFA01-85-C-0013) signed for $500,000.

o May 1, 1985 — First NAS committee meeting.

o June 14, 1985 — Public meeting by NAS for interested parties to provide data, other information, and views to committee.

o April 8, 1986 — Four-month extension to the FAA/NAS contract (originally April 11, 1986) was granted at the request of NAS, with concurrence of the Committee on Commerce, Science, and Transportation.

o August 12, 1986 — NAS transmitted its report, "The Airliner Cabin Environment--Air Quality and Safety," to the FAA.

o August 13, 1986 — Press conference by NAS and public release of the report.

o September 19, 1986 — Hearing before Senate Committee on Commerce, Science, and Transportation, Subcommittee on Aviation on NAS report.

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NAS Recommendation Number 1--"The Committee believes that the health effects associated with air travel should be within the purview of a Federal agency."

Comments

The Committee found that no Federal agency has direct responsibility for dealing with health effects associated with air travel.

During preparation of the report, the NAS Committee was informed that the FAA had no responsibility for the occupational health of the cabin crew. Subsequent to the issuance of the report, FAA reviewed its responsibilities regarding occupational health. The FAA Chief Counsel determined that FAA has the authority to regulate the health aspects of an aircraft in operation. Thus, this recommendation stems, to some extent, from an incomplete picture of Federal authority by the Committee.

In fact, the Department does have authority through FAA and the Office of the Secretary covering both crew and passenger health. FAA's authority is discussed at length in Appendix 2.

In addition to FAA's authority for passenger health, the Office of the Secretary is required by law to ensure that airlines provide "safe and adequate service." The courts have upheld the existing regulation of smoking in cabins under this authority. As other cabin air quality problems could reasonably fall within the statutory requirement to provide safe and adequate service, we believe that the Secretary has the authority to issue rules to protect passenger health.

We also note that the following Federal entities have health responsibilities relating to limited aspects of the cabin environment:

a. Department of Health and Human Services (Food and Drug Administration):

Responsible for ensuring that the food, water, and waste systems on board airliners are safe and that the food that is boarded is not hazardous to health.

b. Department of Health and Human Services (Center for Disease Control):

Responsible for identifying and stemming epidemics.

c. Department of Agriculture:

Responsible for the inspection of food on in-bound flights from foreign points.

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The responsibilities of these agencies do not conflict with those of DOT. Within each of these organizations, responsibility for air cabin health falls within the framework of their overall mandate.

Planned Action

Given the broad range of DOT authority over passenger and crew health, as well as the expertise and the nature of the missions of the other agencies which have health responsibilities, we see no reason to consolidate or transfer any authority. We recommend retention of the present system of health responsibilities.

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NAS Recommendation Number 2--"The Committee found that, if the lowest rate of ventilation permitted by current equipment design were used under conditions of full or nearly full passenger loads, the resulting ventilation rate would be at the minimum determined to provide acceptable air quality when smoking is not permitted and other contaminant sources are not present. In the absence of sources of contamination, this rate does not constitute a health hazard."

Comments

This was a major finding of the Committee. This conclusion has a bearing on the recommendations cited by the Committee for further study of cabin air quality, the implementation of a data collection program that measures airflow in airplane cabins, and the proposed ban on smoking.

Planned Action

This was a finding of the committee and requires no action.

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NAS Recommendation Number 3--"A data collection program that measures airflow and contamination in airplane cabins should be implemented."

Comments

In Chapter 7, "Desirability and Feasibility of Additional Data Collection," the Committee lists the principal air quality problems on aircraft that need to be evaluated. The Committee also states that ventilation rate and cabin pressure are the controlling factors for cabin air quality and concludes that actual ventilation rates should be measured under routine flight conditions in all types of commercial aircraft. The Committee then states, "If significant variations are found in an initial study, continual monitoring should be instituted."

However, the Committee also concluded that the available air quality data are insufficient to make such assumptions. This conclusion was the basis for Recommendation Number 19 that FAA establish a program to measure the various environmental factors that determine the quality of cabin air and base continual monitoring upon the results of the initial study.

Planned Action

Discussions, which began in May 1984, will be renewed with the Environmental Protection Agency. A cooperative effort will be made to determine the feasibility of initiating a practical program for making cabin air quality measurements on board airplanes during typical operations. Contaminants from all sources will be considered as a major item in the conception of this program. Another topic that will be included in these discussions is whether the many variables of flight can be monitored or controlled to correlate test results from different flight conditions.

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NAS Recommendation Number 4--"The FAA standard (on carbon dioxide) is much higher than standards for other confined environments. The Committee recommends that the FAA review its carbon dioxide standard."

Comments

The carbon dioxide standard is contained 14 CFR 25.831. The FAA has reviewed the standard and found that rule originated from Civil Air Regulation 4b.371 and that there is no clear explanation why the 3 percent concentration limit was adopted.

The 3 percent concentration limit of carbon dioxide allowed in section 25.831 is double any temporary concentration value allowed by any industrial carbon dioxide concentration short-term exposure limit, and there are no time values associated with the FAA limit.

For the normal operation condition, a more appropriate approach might be to establish a ventilation rate per passenger rather than to set a specific carbon dioxide concentration limit, because there is a correlation between ventilation, passenger load, and carbon dioxide levels.

Planned Action

The FAA will review the need to establish either ventilation limits, reduced carbon dioxide limits, or both. After the review, any needed rule change and advisory material will be proposed.

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NAS Recommendation Number 5--"Therefore, the Committee suggests that FAA carry out a carefully designed program to ensure that cabin ozone concentrations comply with Department of Transportation regulations."

Comments

The Committee, in its report, acknowledges that the FAA has regulations to control ozone in the aircraft cabin air, however, could find no documentation of the effectiveness of the various methods being used by the airlines to control ozone. On page 119 of the report, under RECOMMENDATIONS, it states that in 1978-1979, FAA monitored ozone concentration limits.

Based on the results of those studies, regulations were adopted that were published January 21, 1980, and became effective February 20, 1981.

The Committee further stated that "...because catalytic converters are subject to contamination and loss of efficiency, it is suggested that FAA establish policies for periodic removal and testing, so that the effective life of these units can be established. A program of monitoring is needed to establish compliance with the existing standard and to determine whether the catalytic converters are operating normally and effectively. These data should be maintained in such a manner that they can be used for reference on passenger and crew exposures to ozone and to document the concentrations of ozone."

On August 28, 1984, the FAA issued a general notice (GENOT N8320.300) to determine the status of air carrier compliance with 14 CFR 121.578, Cabin Ozone Concentration. As of November 2, 1984, information had been received on 63 operators. Fifty-two operators were complying with the rule by following operating procedures or flying at levels below 27,000 feet. Eleven operators had installed catalytic converters and had various programs to replace and/or test the efficiency of the converters.

With respect to a program to monitor ozone, the FAA conducted tests during 1978 to 1979. A total of 157 flights were sampled over a 2-year period. At that time, the FAA estimated that the number of samples would have to be increased by an order of magnitude to be statistically meaningful.

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Planned Action

The FAA will issue biennial action notices, which have replaced GENOT's, requiring FAA inspectors to report on the present status of all U.S. air carriers' compliance with the existing ozone regulations, results of catalytic converter efficiency tests, and a listing of any validated complaints related to cabin ozone. The responses to the action notices will be summarized and published. Identified deficiencies will be corrected.

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NAS Recommendation Number 6--"The Committee recommends a ban on smoking on all domestic commercial flights."

Comments

The Committee cited four reasons for banning smoking on all domestic commercial flights:

a. to lessen irritation and discomfort to passengers and crew;

b. to reduce potential health hazards to cabin crew associated with environmental tobacco smoke (ETS);

c. to eliminate the possibility of fires caused by cigarettes; and

d. to bring the cabin air into line with established standards for other closed environments.

The Committee noted that the irritation reported by passengers--which includes eye irritation, headaches, and coughing, among others—all affect the general health and welfare of the passengers and crew.

The Committee found ETS to be a hazardous substance having components that are toxic, carcinogenic or cocarcinogenic. The Committee found that there is a positive association between lung cancer and exposure to ETS.

While DOT agrees that the banning of smoking could marginally reduce the possibility of fires caused by cigarette smoking, we believe that recent actions by the Department, including the use of fire-blocking materials in cabins and smoke detectors in lavatories, eliminate nearly all risks of fire due to smoking.

The Committee's recommendation to ban smoking in order to bring the air quality in line with established standards for other closed environments is based on limited measurements. These data show that, in many cases, the air quality of cabin environments violate the EPA 24-hour particulate standard.

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Planned Action

While we recognize that exposure to ETS could be viewed as a problem by some crew and passengers, we believe that further study is needed before the Department can propose a definitive response to this recommendation.

Issues that require further consideration include the following:

- Further review of the health effects of ETS, including review of the studies and reports completed since the NAS study.
- Further review of the concentration and distribution of pollutants for various aircraft types.
- The effectiveness and economic effects of possible technological solutions such as modifications to aircraft ventilation systems or procedures.
- Further consideration of possible application of any proposed solutions to international, as well as domestic, flights.

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NAS Recommendation Number 7--"Because a likelihood of occurrence of epidemic disease when forced-air ventilation is not available on the ground has been demonstrated, the Committee recommends that a regulation be established that requires removal of passengers from an airplane within 30 minutes or less after a ventilation failure or shutdown on the ground and maintenance of full ventilation whenever onboard or ground air-conditioning is available."

Comments

As the Committee stated in its report, there has only been one documented and confirmed incident of a communicable disease spreading within an airplane on which ventilation was inoperative while repairs were attempted. The turbojet airplane had aborted takeoff departure from Homer, Alaska, due to loss of thrust in one engine. The nature of the repairs necessitated removal of the ground ventilation system for the cabin interior. Passengers were offered a choice of deplaning to the somewhat rudimentary terminal or remaining on board the airplane. Either choice apparently offered approximately the same degree of protection from the elements. The outside temperature was slightly below freezing. Some of the passengers did elect to wait out the delay in the terminal.

One passenger, who had boarded at Homer and elected to remain on board, was afflicted with early stages of influenza. All of the passengers who remained on board were afflicted with influenza in varying degrees of severity within a few days. None of the passengers from the terminal contracted the illness during the resumed flight to Kodiak on a substitute, normally ventilated airplane. The conclusion was that the illness had been transmitted throughout the grounded airplane due to lack of ventilation.

DOT agrees that confinement in an unventilated enclosure (room, car, bus, etc.) will facilitate spread of epidemic disease. Because the occurrence of complete ventilation cessation on passenger-laden airplanes is extremely rare, and as in the Homer case, unavoidable, we do not believe that regulatory action is necessary. Unlike Homer, the cessation is usually very brief. The duration of such instances is likely to be less than 30 minutes.

Planned Action

While the risk of occurrence of complete ventilation cessation on passenger-laden airplanes is extremely low, we believe that there may be value in bringing this concern to the attention of the air carriers. The FAA will advise air carriers of the need to deplane passengers, if possible, after 30 minutes without ventilation.

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NAS Recommendation Number 8--"The Committee also recommends that maximal airflow be used with full passenger complements to decrease the potential for microbial exposure and that recirculated air be filtered (to remove particles larger than 2-3 um) to reduce microbial aerosol concentrations."

Comments

Maximal airflow is used on a regular basis today in airline operations. During the past fuel crisis, there was a common practice of shutting off air-conditioning packs to conserve much needed fuel. With all of the air-conditioning packs operating, the current fleet of airplanes exceeds or equals The American Society of Heating, Refrigerating, and Airconditioning Engineers, Inc. (ASHRAE) Standard 62-1981 of 7 CFM for auditoriums, theaters, and other nonsmoking spectator areas.

Older jet transports were designed to deliver a cabin fresh air ventilation rate of 15 to 20 cubic feet per minute (CFM) per passenger. Newer airplanes, such as the Boeing 757 and 767, were certificated with a mixture of air consisting of approximately 10 to 15 CFM of fresh air and 5 to 10 CFM of recirculated air. All of the newer aircraft incorporate some form of filtration for the recirculated air and are already being delivered with the 2-3 um (one-millionth of a meter) or better hospital-type filters.

DOT does not have a data base of reports of the spread of illness or disease within the airplane cabins that are ventilated by environmental control systems operating at design levels. DOT agrees with the Committee's statement on page 159 of its report that "Microbial concentrations have not been measured in aircraft, and therefore accurate risk assessments cannot be made." As discussed under Recommendation Number 19, there is merit in making cabin air quality measurements, including concentrations of microbial aerosols. These measurements would be taken with different levels of cabin airflow to establish a correlation between microbial concentrations and the amount of airflow. Once this correlation is established, the health effects of marginal airflow can be established.

Planned Action

The program for making cabin air quality measurements identified under Recommendation 19 will include microbial aerosol concentrations at different cabin airflows. Whether or not maximal airflow is required at all times will be evaluated at the conclusion of this measurement program.

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NAS Recommendation Number 9--"The Committee concluded that current pressurization criteria and regulations are generally adequate to protect the traveling public. However, the medical profession should use a more efficient system to warn those with existing medical conditions who are more susceptible to changes in pressure or to long exposure to low pressure that there might be some hazard to their health."

Comments

DOT concurs with the Committee's conclusion that the current pressurization criteria and regulations are adequate to protect the traveling public with the exception of a very small percentage of those with preexisting medical conditions that increase their susceptibility to changes in the partial pressure of oxygen and cabin pressurization. Because of the possibility of an in-flight medical emergency secondary to a preexisting condition, the FAA requires the availability of supplemental oxygen for first-aid use. Crewmember training in the care of passenger's conditions that may result secondary to the in-flight environment is also required. The responsibility of warning patients with such medical conditions is considered to be that of the patient's physician. The FAA has an ongoing program to educate that segment of the medical profession with which it has direct contact (FAA-designated Aviation Medical Examiners), and it also encourages the publication of articles concerning aviation medicine in medical journals as an adjunct to the educative effort.

Planned Action

The FAA will continue its ongoing effort to provide aviation medical information to the members of the medical profession to enhance their ability to advise those patients at risk concerning possible hazards secondary to the in-flight environment. In addition to direct contact with FAA-designated Aviation Medical Examiners, the FAA will continue to encourage publication of such information in medical journals. The FAA will ask the American Medical Association to republish the excellent article "Medical Aspects of Transportation Aboard Commercial Aircraft" (Journal of the American Medical Association, February 19, 1982, Volume 247, Number 7) (Appendix 3).

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NAS Recommendation Number 10--"FAA should consider rule-making that restricts exposure [to cosmic radiation] of pregnant flight crew and cabin crew members. In addition, FAA should investigate total radiation exposure of flight crew and cabin crew members through the use of a statistical sample of full-time employees and should require airlines to provide precautionary information to their flight attendants about radiation exposure."

Comments

The FAA has been interested in the effects of increased natural ionizing radiation since the early planning stages of civil supersonic transportation. Because of research efforts in this area, much data have been gathered pertaining to the extent and effects of radiation at supersonic and subsonic aircraft flight altitudes. Since the initiation of supersonic passenger flight on the Concorde, actual radiation exposure has been monitored during flight and documented. Operational experience over the past 8 years with Concorde aircraft operated by Air France and British Airways has proven conclusively that the risk of exposure to radiation to passengers and crewmembers is very low. Since the amount of radiation is related to altitude, there is even less exposure in the operation of subsonic aircraft.

Galactic radiation is predictable in relation to altitude and latitude. The radiation from solar flares is not predictable, but subsequent exposure can be minimized once the event occurs by avoiding high altitude, high-latitude flights.

Crewmembers at risk of exceeding the 500 millirems annual limit represent only a very small percentage of those engaged in the air carrier industry. Since the radiation environment at flight altitudes is well defined and reasonably predictable, the expense and burden of mandatory radiation controls and monitoring are not realistic.

Air carrier crewmembers, in most cases, have the unique opportunity to vary their occupational scheduling as related to high-altitude, high-latitude flights. This is particularly significant in the case of crewmembers who are pregnant or may have been exposed to solar flare activity. With appropriate advisory information and management cooperation, crewmembers will have the option to limit their exposure to cosmic radiation by discrete scheduling within domestic and flag route systems. The FAA has considered rulemaking and finds that there is insufficient evidence to justify it. Appropriate scheduling by flight attendants can eliminate the threat perceived by the NAS.

Planned Action

The FAA is currently developing advisory information to promote radiation safety through educational initiatives for use by the air carrier industry. This information will be distributed, and with management cooperation, crewmembers concerned with radiation exposure will be able to limit such by scheduling within domestic and flag route systems.

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NAS Recommendation Number 11--"The Committee approves of current efforts to base passenger safety briefings and written materials on empirical testing of comprehension and retention."

NAS Recommendation Number 12--"The Committee suggests that FAA or appropriate industry organizations consider the advisability of developing an empirical research program to examine passenger response to safety instructions under routine and emergency conditions and revise them as appropriate. Consideration should be given to running some quizzes during a flight to see, for example, what proportion of passengers have retained the key features of the safety briefing."

Comments

Air carriers are encouraged to make the required preflight and optional in-flight and prelanding briefings understandable and motivational in nature. The guidance concerning these briefings is contained in Advisory Circular 121-24 (Appendix 4), which is designed to standardize and improve the safety information presented to passengers by the airline industry. Guidance is provided concerning the preparation of comprehensive oral briefings and written passenger briefing cards. The accepted practice of both the FAA and the airline industry is that briefings concerning emergency actions should not create passenger apprehension or inspire unwarranted actions by the passengers. AC 121-4 is currently being revised to include the latest state-of-the-art in briefing techniques and technological advances.

Attentive passengers who have received the preflight briefings could "pass" quizzes given during nonstressful flight situations, however, that does not necessarily mean that they will take the correct action in a given emergency situation. Cabin attendants, who have received hands-on, simulated emergency training, including techniques for disoriented and panicky crowd control, have the dominant role in emergency situations over passengers who have received only indoctrinational type briefings. The emergency procedures for passengers are, therefore, relatively passive (i.e., follow instructions given for their individual protection) while trained crewmembers implement procedures and deploy any required equipment necessary to assure the continued safe flight and landing of the airplane and the safety of the passengers.

The FAA has considered the advisability of developing an empirical research program as recommended and finds no basis in the NAS report or elsewhere to justify the research. The FAA is deeply interested in suggested improvements for briefing techniques from the airline industry, but requiring a quiz data base is inappropriate.

Planned Action

The FAA will continue to update AC 121-24 as necessary.

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NAS Recommendation Number 13--"The Committee recommends that FAA require that information on proper response to fire emergencies be included in oral and written passenger safety information."

Comments

Federal Aviation Regulations 14 CFR 121.571 and 135.117 contain the requirements for briefing air carrier passengers. The guidance for operators of airplanes having a seating capacity of more than 30 passengers and for FAA inspectors is contained in Advisory Circular 121-24.

AC 121-24 was designed to standardize and improve the safety information presented to passengers by the airline industry. Guidance is provided in the AC concerning the preparation of comprehensive oral briefings and written passenger briefing cards. The accepted practice of both the FAA and the airline industry is that briefings concerning emergency actions should not create passenger apprehension or inspire unwarranted actions by passengers. The emergency procedures for passengers are, therefore, relatively passive (i.e., follow instructions given for their individual protection) while trained crewmembers implement procedures and deploy any required equipment necessary to assure the continued safe flight and landing of the airplane and the safety of the passengers.

Recent occurrences of unwarranted passenger-initiated emergency evacuations have caused concern that preflight briefings may in some cases motivate some people to act independently and unnecessarily. When trained cabin crewmembers are present, the best course of action is for the passengers to remain seated while specialized crew action is accomplished to alleviate smoke or fire.

At the present time, the only special equipment or procedures for passenger usage are for airplanes of 19 passengers or less, which have no cabin crew and are operated under 14 CFR Part 135. In these operations, the pilot in command must comply with 14 CFR 135.117 and adequately brief passengers to accomplish emergency procedures in the event of cabin fire, to assure continued safe flight and landing and subsequent evacuation.

Planned Action

The FAA will continue the present practice.

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NAS Recommendation Number 14--"The Committee feels that continuing research (fire safety) is also needed in materials development."

Comments

As the basis for this recommendation, the Committee states on page 10 of its report, "In general, the FAA program on flammability testing is excellent, and its research efforts to improve testing methods are appropriate and valuable. The recently issued FAA flammability standards for seat cushions and cargo compartment liners will reduce in-flight and postcrash fire hazards." "...Although FAA standards are met by currently available materials, other materials exist that, with further development, would far exceed current standards and would provide substantially increased fire protection in aircraft."

Continuing research is needed in the development of improved fire-safe materials. The FAA's part in this research generally entails the analysis of fire-safety problems and the development and validation of practical small-scale test equipment that can be used to measure reliably the relative fire-safety characteristics of materials. Using this test equipment, the FAA cooperates with material producers, interior fabricators, aircraft manufacturers, and airlines in the development and adaptation of new materials which have a high level of fire safety as well as durability, cleanability, appearance, and other practical characteristics necessary for economically competitive materials.

Recent examples of this highly-successful, cooperative approach between FAA and industry include the establishment of new standards for heat-resistant evacuation slide materials, cargo compartment fire barrier liner materials, fire-safe seat cushion materials, and cabin wall and ceiling panel materials.

Planned Action

The FAA plans to continue this approach in the development of improved materials and believes that this action is responsive to the Committee's recommendation.

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NAS Recommendation Number 15--"The Committee noted that current emergency procedures for smoke removal recommend that the cabin be depressurized to 10,000 ft. This procedure is ineffective and should be discontinued."

Comments

The Committee on Airliner Cabin Air Quality presented the following reasoning: "All procedures the Committee reviewed specified increasing cabin altitude to 10,000 ft. to increase ventilation. Although that will increase the volume of air flowing through the cabin, the lower pressure will also increase the volume of smoke produced by a given fire, and there would be little or no reduction in smoke concentration. Any reduction in burning rate due to the decrease in partial pressure of oxygen in the cabin is insignificant."

Mass airflow is generally controlled by the pressurization schedule and the characteristics of the bleed air system. The significance of combined changes in air density, air velocity, and airmass flow on burning rates or smoke production is unknown. There may be too many unpredictable variables to draw any justifiable conclusions.

On September 29, 1986, the FAA issued Advisory Circular (AC) 25-9, Smoke Detection, Penetration, Evacuation Tests and Related Flight Manual Emergency Procedures (Appendix 5). This new AC provides guidelines for the conduct of certification tests relating to smoke detection, penetration, and evacuation procedures. The AC also provides guidelines to evaluate related airplane flight manual procedures.

AC 25-9 has been reviewed, and it has been determined that paragraph 5b is too general relative to smoke evacuation procedures. The paragraph should be more specific and note the limitations of smoke procedures.

Planned Action

The FAA will obtain and review additional information that is known to address the smoke evacuation issue. This will include a Lockheed study that investigated decompression as a means to control or extinguish a fire, pertinent information retained by the cognizant aircraft certification offices, and any additional information from the NAS. NTSB in-flight smoke and fire accident/incident reports will also be reviewed. Based on this review, the FAA will determine the effectiveness of the current procedures for smoke removal.

AC 25-9 will be revised to be more specific relative to decompression procedures and include any limitations on their use, or other appropriate actions as determined to be necessary will be taken.

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NAS Recommendation Number 16--"However, there are generally more crew members than fire extinguishers, and the Committee recommends that FAA review the proposed rule on protective breathing devices for crew members to ascertain the desirability of supplying such equipment for all crew members, rather than limiting it to the persons expected to be involved in firefighting. In addition, the Committee suggests further evaluation of the potential of emergency breathing equipment for all cabin crew members to improve safe and expeditious evacuation of passengers in fire emergencies."

Comments

The NAS report made note of current FAA Notice of Proposed Rulemaking (NPRM) 85-17 (Appendix 6) concerning protective breathing equipment (PBE) for crewmembers. NPRM 85-17 was published in the Federal Register for public comment on October 10, 1985, with a closing date of February 10, 1986. The proposed rulemaking stemmed from recommendations made by the National Transportation Safety Board concerning accidents caused by in-flight fires. Therefore, the thrust of the proposed rule is to provide protection for persons directly involved in addressing cabin smoke and fire while in flight.

The recommendation to evaluate the potential of emergency breathing equipment for all cabin crew members to improve safe and expeditious evacuation of passengers will not be addressed as part of the above discussed rulemaking but will be addressed as part of Recommendation Number 17 to reexamine passenger protective breathing devices.

The fighting of a fire presents significant workload demands that dictate the design of the protective breathing devices provided for that purpose. When the cabin crewmembers are not fighting an in-flight fire, their needs for protective breathing equipment would be similar to those of the passengers.

Planned Action

The evaluation of the potential of emergency breathing equipment for all cabin crewmembers to improve the safe and expeditious evacuation of passengers in fire emergencies will be conducted in conjunction with the planned action on Recommendation Number 17.

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NAS Recommendation Number 17--"The Committee recommends that FAA re-examine passenger protective breathing devices and consider requiring that such equipment be available in case of in-flight and postcrash fires."

Comments

The FAA concurs with the Committee's recommendation to restudy the issue based on current technological developments throughout the world. The FAA is currently reviewing this new technology with representatives of the Civil Airworthiness Authorities of Canada, France, and the United Kingdom.

On July 29, 1986, the FAA received a letter from the United Kingdom's Civil Airworthiness Authority, including a specification for passenger protective breathing equipment (PBE) for both in-flight and postcrash fires. The Canadians have also been conducting research on passenger PBE as a result of the Cincinnati accident. The Canadian's work is referenced in the NAS report. In addition, the FAA learned that France is conducting testing in passenger PBE.

A meeting was held between representatives of the four countries in Redhill, England, September 29, 30, and October 1, 1986, to discuss research and development (R&D) and to develop a consolidated, cooperative plan to conduct the necessary R&D to reinvestigate the technical safety merits of PBE.

The general consensus of the meeting was that two scenarios need to be investigated, in-flight and postcrash. The representatives decided that before any regulatory action could be contemplated, it would be necessary to identify the need, establish candidates' scenarios, identify test objectives, and establish detailed pass/fail criteria.

Planned Action

This joint international effort on passenger protective breathing equipment will continue. Several meetings have been held during the last 6 months. The groups' objective is to perform a worldwide accident and incident analysis to fully summarize fire events and human factors, in order to evaluate the safety potential of individual passenger protective breathing devices under different fire scenarios. The group will also begin development procedures for testing the devices.

A meeting will be held in May 1987 between the four nations to decide on further activity. Once this international effort is completed, the FAA will assess the need for regulatory action.

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NAS Recommendation Number 18--"The Committee was charged with performing a comparison of foreign industry practices, regulations, and standards, and has gathered relevant information applicable to the issues addressed in this study. Although some differences from those in the United States have been noted, they do not appear to be significant. The Committee feels that greater effort along these lines is not warranted."

Planned Action

This recommendation requires does not require DOT action.

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NAS Recommendation Number 19--"The Committee therefore recommends that FAA establish a program for the systematic measurement, by unbiased independent groups, of the concentrations of carbon monoxide, respirable suspended particles, microbial aerosols, and ozone and the measurement of actual ventilation rates, cabin pressures, and cosmic radiation on a representative sample of routine commercial flights. These findings should be subjected to peer review."

Comments

(NOTE: The term "program" as used in this recommendation is interpreted to mean a one-time study.)

In 1970 and 1971, FAA and the U.S. Public Health Service (PHS) conducted a study of the health aspects of smoking in passenger-carrying aircraft. The study involved the measurement of tobacco combustion by-products, including carbon monoxide, smoke particulates, aromatic hydrocarbons, aldehydes, and ketones on 20 military airlift command international flights and 14 domestic civilian flights. The results revealed very low levels of each contaminant measured, much lower than those recommended in occupational and environmental air quality standards. This was the earliest effort to evaluate air contaminants in passenger-carrying aircraft.

The Committee cited other tests that were performed by airlines and individuals, including "measurements of opportunity," made by some of the Committee members. In most instances, carbon monoxide and respirable suspended particles of environmental tobacco smoke were measured. Carbon monoxide levels far below ambient and occupational standards were reported. There are no established standards for smoke particles.

The levels of microbial aerosols (e.g., viruses, bacteria, molds, and fungi) that may exist in airliner cabins were not considered in either the FAA/PHS 1970-71 study or in subsequent tests. Recent indoor air pollution investigations have revealed that a wide variety of microbial contaminants are commonly found in occupied, enclosed spaces such as modern office buildings. Presumably, such would be the case in any closed environment where people congregate, and passenger-carrying aircraft would be no exception. Despite the fact that there are no standards limiting the kinds and concentrations of microbes in such places, the Committee stated that "...in view of the degree of expressed concern about microbial contamination in aircraft and the possibility that serious acute health effects could result from such contamination, it is important to collect baseline data on background concentrations of microbial aerosols during normal flight conditions."

The FAA/PHS 1970-71 study of tobacco smoke contamination in aircraft has been labeled by some as "inadequate." However, the limited data that have been collected in subsequent less-structured testing, prior to the NAS study, have done little to refute the conclusion reached in the early study, i.e., that, based on environmental levels and expected dose-response relationships of contaminants, tobacco combustion products do not represent a hazard to the nonsmoking passengers.

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Data obtained with the computer model developed by the Committee were found to be supportive of the limited empirical data. All of the data suggested that "...cabin air is probably no worse than air in many other confined environments." This conclusion by the Committee was based on data that did not consider the potential for microbial aerosol contamination.

The technology for measurement of the environmental factors associated with cabin air quality has made tremendous strides since the 1970-71 FAA/PHS study. Also, the experimental designs used in that and the other studies cited by the Committee were probably less than adequate. Designing and conducting new studies using more sophisticated investigative techniques could increase the level of confidence in the measurements obtained.

Planned Action

Discussions, which began in May 1984, with the Environmental Protection Agency will be renewed regarding the advisability and feasibility of initiating a practical program for making cabin air quality measurements on board airplanes during typical operations.

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NAS Recommendation Number 20—"The Committee recommends that FAA establish a program to monitor selected health effects on airliner crews."

Comments

Acting under its responsibility for the occupational safety or health of aircraft crewmembers, the FAA has issued numerous regulations directly affecting the workplace of pilots, flight engineers, cabin attendants, and other persons whose workplaces are on aircraft in operation. These regulations cover aircraft performance and structural integrity, safety equipment for emergency ditching and evacuation, fire protection, protective breathing equipment, and emergency exits. Other regulations affecting the crewmember workplace have been issued with respect to cockpit lighting, crewmember seat belts, toxicity and other characteristics of materials in the crewmember workplace, and other environmental factors affecting that workplace, including noise reduction, smoke evacuation, ventilation, heating, and pressurization. Maximum hours of duty and duty aloft for air carrier flight crewmembers are also regulated, as is the protection of crewmembers from radioactive and other hazardous materials.

Although the FAA has no reporting requirement for crewmember disease that may be occupationally related, there are data available on in-flight crewmember injuries. The FAA also requires periodic physical examination on flight crewmembers; however, there is no such requirement for flight attendant crewmembers. There is no conclusive evidence of increased incidence of disease related to crewmember duties.

The type of monitoring program the NAS report recommends would attempt to identify health effects of the working environments on cabin crewmembers. In making the recommendation to examine rates of spontaneous abortion and birth defects among cabin crewmembers, the NAS report identifies the difficulty of studying such a complex, phenomena where it is difficult to separate the effects of the cabin environment from other environmental or hereditary effects. The NAS report indicates difficulties in interpreting the results that will occur and recommends that "a feasibility study be undertaken to determine whether these conditions can be met." The FAA will discuss with the National Institute of Occupational Safety and Health (NIOSH) the feasibility of establishing a program to monitor selected, health effects on airline crews.

Planned Action

The FAA will continue its efforts to ensure the occupational health and safety of crewmembers in response to established needs. The FAA will discuss with NIOSH the feasibility of establishing a program to monitor selected health effects on airline crews.

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NAS Recommendation Number 21--"The Committee recommends that FAA collect these data (uses of recently mandated medical kits) in such a way as to permit comparison of onboard incidents with those in other settings."

Comments

FAR Amendment Numbers 11-29 and 121-188 (Appendix 7), which contain the requirement for the carriage of emergency medical equipment on air carrier aircraft, became effective August 1, 1986. New 14 CFR 121.715, included in these amendments, requires that for a period of 24 months, each certificate holder will maintain records on each medical emergency occurring during flight time resulting in the use of the emergency medical equipment, diversion of the aircraft, or death of a passenger or crewmember. These records will include how the medical kit was used, by whom, and the outcome of the medical emergency. This information will be submitted to the FAA within 30 days after the end of each 12-month period during the 24 months of required reporting. This existing data collection program is fully responsive to the NAS recommendation.

Planned Action

The FAA will use the data required by 14 CFR 121.715 to determine future need for the carriage of emergency medical equipment on air carrier aircraft. This data will be available for use in studies comparing medical incidents on board air carrier aircraft with those in other settings. The FAA will not undertake these comparative studies since it does not have the authority. Such studies would be inappropriate for the FAA.

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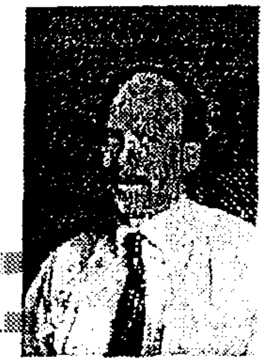

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Research Interests:

Throughout our lives we are exposed to environmental contaminants present in air, water, food and soil. The thrust of our research activities is the assessment of population exposures that occur in homes, offices, schools, and during transit as well as i n the outdoor environment. Most of these assessment studies are carried out in conjunction with epidemiologic investigations.

Our studies as well as others reveal that we spend 65% of our time in our residences, 25% in some other indoor environment, 5-7% in transit, and usually less than 5% of our time are we actually outdoors. The contaminant levels encountered in these indoor environments are important contributors to exposure, discomfort, irritation and health effects. Although we assess the effects of pollutants of outdoor origin (ozone, acidic particles, PCBs) we are particularly interested in pollutants of indoor origin (fungi, dust mites, nitrogen dioxide, tobacco smoke, lead, asbestos, volatile organic compounds, formaldehyde, radon, and others). Concentrations of these pollutants can reach levels many times greater than outdoor levels. NO2 levels in hockey rinks can exceed 1 ppm while homes with unvented gas cooking might vary between 20 and 100 ppb. In homes across North American, the presence of molds and mildew has been associated with substantial increases in upper and lower respiratory symptoms. Volatile-comp onents in gasoline can be 6 to 10 times higher inside a passenger car during rush hour traffic than values measured at standard urban monitoring sites.

Careful assessment of human exposures has enhanced our epidemiology by impr ving upon descriptor and surrogate indicator variables. Exposure measurements have been extremely helpful in identifying plausible associations in sick building investigations. As we continue an EPA-sponsored National Human Exposure Assessment study, we will be revealing behavioral, housing and geographic factors that lead to high exposures, body burdens and health risk to pesticides (chlorpyrifos, diazinon), metals (lead, arsen ic), and volatile organic acids (benzene, toluene and chloroform). New research efforts return us to issues of ozone exposure. More than 200 young children in Los Angeles are wearing personal ozone monitors developed here at Harvard School of Public Hea lth by Professor Petros Koutrakis. In a related study, we are investigating the chemistry of ozone as it degrades on the internal surfaces of air ducts in buildings and other surface materials in homes.

Selected Publications:

Spengler JD, Schwab M, Ryan PB, Billick I, Colome S, Wilson AL, and Becker E. Personal exposure to nitrogen dioxide in the Los Angeles Basin. *Journal of the Air and Waste Management Association*, 1994, 44:39-47.

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DEPOSITION EXHIBIT
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Spengler JD, Burge H, Dumyahn T, Dahlstrom C, Muilenberg M, Milton D, Ludwig S, and Weker R. *Aircraft Cabin Environmental Survey, An Executive Summary,* May 21, 1994.

Spengler J, Neas L, Nakai S, Dockery D, Speizer F, Ware J, and Raizenne M. Respiratory symptoms and housing characteristics. *Indoor Air*, 1994, 4:72-82.

Spengler J and McCarthy J. Indoor air quality in hospitals: not just another building. *NATO Workshop Proceedings*, Milan, Italy, September 1995.

Spengler J and Ford T. *From the Environmentally-Challenged City to the Ecological City.* Presented at the Conference on Cities in North America, New York, NY, June 14-16, 1995.

Administrative questions and faculty requests to amend this Web page should be referred to Deane Eastwood, Office for Academic Affairs, at deastwoo@sph.harvard.edu

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